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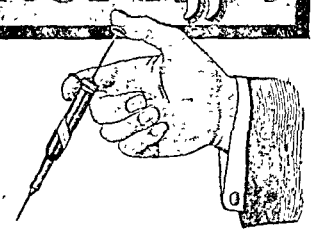

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ORIGINAL ARTICLES.

THE EMMANUEL MOVEMENT.

ITS PRETENSIONS; ITS PRACTICE; ITS DANGERS.

By John K. Mitchell, M.D.,

PHYSICIAN TO THE ORTHOPEDIC HOSPITAL AND INFIRMARY FOR NERVOUS DISEASES, PHILADELPHIA.

Even before the Emmanuel movement began, the doctors had been hearing so much about mental healing that we were growing a little tired of being instructed and exhorted by the laity; then came the flood of literature let loose by the new agitation, and our interest was renewed until we discovered that we were only being offered an old, well-tried, and respectable remedy (with a few new adornments, to be sure) under a novel and flashy name—psycho-therapy—soul-treatment! It looked a little like Christian Science, it sounded rather like "mind-cure," it had some elements of the "faith-cure," but every combination of psyche and psycho was fashionable; it seized the fancy, and here it is, so well established a phrase that one must not waste time on it except to cast a doubt on its scientific accuracy.

Let us say at once that there can be no question that the Emmanuel movement has done good, and that in the hands of cool-headed men of wide knowledge of the world, deep versed in human nature, and possessed of an unusual combination of caution, courage, and acute judgment, it may do more. Its important service in the end will be to have called attention anew to the fact, already always and everywhere held and taught by thoughtful doctors, that the mind and the body cannot be treated independently of one another.

Some of the more direct and immediate benefits received by—are they to be called its patients?—we have heard of. There are yet certain doubts to be expressed on the statistics of these cures. Let us admit them for the present in evidence and agree that much has been accomplished. Even then we ought not to approve, as a whole, a method which proposes that the clergyman shall call in the physician, not to direct or even aid in the treatment of the case, but to relieve him of some responsibility by settling whether the sufferer shall be turned over to the spiritual arm. We hold our profession as seriously in honor as the clergy do theirs, and it is not asking too much that they should admit that both callings have in this discussion one object in view: the welfare of those whose health, whose happiness—yes—whose souls are in our hands. There need be, there should be, no jealousy—but the tone used by some of the supporters of the Emmanuel movement toward our profession will bear improvement. It is childish for them nowadays to rake up ancient slurs on physicians, to talk about doctors being materialists or atheists, and such medieval gibes as should have been long barred by a statute of limitation on old jokes that never were good ones.

This is no place for consideration of whether doctors are mostly good Christians or religious men, but it may be safely asserted that "idealist" rather than "materialist" would be the word fitly to describe the largest number of doctors to-day Not very long ago Miss Cobbe complained, in an article published in England, that the doctors "were setting up a new priesthood which was to replace the care of the soul by the care of the body." But since some of the clerical champions of soul-healing seem in their zeal almost to have forgotten that souls have bodies, they may well be reminded that the opinion of the best men of our profession has ever been that he was but a poor physician, a mere mender of broken bodies, who was not concerned for the spirit as well as for the flesh. Most earnestly should we insist that the *treatment* of a patient, whether it be surgical, medical, or psychic, should, for the safety of the public, be in the hands of the doctor.

A physician's first duty is to cure his patient. We who are doctors belong to no school, owe allegiance to no medical sect, admit no creed which binds us to a limited system. We stand on one principle and one only. We will use any honest means to heal our patients. The wisdom of the ancients, the science of the moderns the art of the practitioner—we use them all with such minds as we have and such skill as we can acquire by painful years of study and laborious work. Our professional hospitality has turned no man away who offered us any means to this end, whether he were a Jesuit missionary bringing cinchona bark from South America, or an African voodoo-man with an ordeal poison. We listen, we try, we adopt or lay aside. Always the best of our calling—and it is by these we may claim to be judged—have been willing to be taught by

anyone. Of late years the medical man, far from being a reactionary, has been almost too ready to accept the new, often receiving half-fledged and doubtful remedies rather on faith than by works. It cannot, therefore, be said that opposition to the new movement is opposition to an innovation because it is an innovation. Rather is it opposition to the form of *re*novation of an old heresy. What is new in the Emmanuel movement has been forced upon it by medical criticism after a years' exploitation of the plan: that the doctor shall be called in to decide whether he shall gracefully retire and hand over the patient to the—shall we say unlicensed?—practitioner. What is old is that an appeal to the best in man, confidence in the goodness of God, the stimulation of the patient's self-helpfulness, courage and faith, are useful means of aiding those who are in trouble, sickness, or any other adversity. The thoughtful physician has used these means since there were physicians, since the days, in fact, when the functions of the priest and the medicine-man were differentiated. Before that time pure psychotherapy was practised by the ecclesiastical authorities, shaman, voodoo-man, or witch-doctor, who put feathers in their hair, painted their faces, and beat tom-toms to scare away the evil spirits that produced the illness. Further development brought the medical man out of the medicine-man and gave up the treatment of disease by these wholly spiritual and suggestive means as unwise. How far are we to return on our steps now? and shall the specialized physician be replaced again by his primeval ancestor, the combined priest and medicine-man?

Our clerical critics disclaim any intention of finding fault with physicians, and then explain that they only mean that doctors are materialists and materialistically taught, imperfectly educated, and not acquainted with modern science; fifty years behind Europe, and quite unequal to the task of healing nervous patients, which is to be assumed by the ministers, who presumably are fifty years ahead of Europe,[1] in which benighted land treatment of the sick is still strictly a duty of the physicians.

It can scarcely be seriously contended that the clergy in general are better fitted to undertake the cure of mental disease than the doctors in general. I desire to speak of the ministers as I regard them, with every respect for the men, and more for the office. Nevertheless, since we are frankly criticising one another, I must say that in my experience the minister when called in to perform his recognized and desirable functions in aid of patients in spiritual

[1] The Rev. Lyman P. Powell, far the coolest and most level-headed of the Emmanuelists, and author of a book which has some semblance of the scientific and statistical method so lacking in the "official" publications, said at the Episcopal Congress last May that "physicians in America were fifty years behind Europe in this regard" (psychotherapy). "Its success depended on no theory of the subconscious; it put the responsibility on the physician;" exactly—the responsibility, but neither the authority nor the credit for good results. (Reported in The Churchman, May 22, 1909.)

need, has not often been so helpful as one would expect or hope. Of course, we are speaking of his functions as pastor and priest, not as a psychic assistant. It has seemed sometimes as if he were unable to comprehend that the minds of such patients work in contradictory and perverse fashion, and could not be judged by normal standards, or that he must distinguish the normal disturbances due to religious doubts from the self-accusatory mental attitudes of persons ridden by fixed ideas of sin or mere unformulated vague indecisiveness. Even these two latter troubles, though frequently they are heralds of serious mental or brain diseases, may be helped to some extent by strong, simple authoritative reassurance and reenforcement of the courage. It is not from want of good-will that the minister is of so small helpfulness in these instances, but sometimes from want of knowledge of the world of men, and oftener rom inexperience in mental disorders and from lack of training—exactly the defects charged to the medical profession. One does not speak of the failures to help where we all may fail, namely, in the cases of those patients who, whether they consult a minister or a doctor, are seeking rather a novel emotional stimulant than the real help which comes only through stern discipline and self-control, two measures very unpopular among neurotics. The causes of this imperfect comprehension and consequent inability to help lie not only in the absence of training in the study of abnormal mental states and, without disrespect be it spoken, the credulity with which the patients' overaccented statements are usually received by the inexperienced, but also in another important and rather contradictory fact—the notion, no less widely held by the clerical profession than by the laity, and not unknown even among medical men, that nervousness in almost any form or expression is an "imaginary" ailment. This attitude on the part of the adviser from whom the patient has every right to anticipate understanding, consolation, and spiritual strengthening has frequently unfortunate results: if the too easy belief give the prevailing tone, the patient, delighted with the prospect of a new and sympathetic auditor, goes on from exaggeration to exaggeration, or if his case be one of genuine need of spiritual comfort, and he finds his trouble regarded as imaginary, his depression is deepened and his spiritual helplessness intensified by the feeling that the one of all others whom he has looked to for comprehension does not understand his state. The authors of *Religion and Medicine* have admitted some of these deficiencies and pointed out themselves the much greater success with which the Roman Catholic priest handles such difficulties, though they attribute this success to the confessional, which is probably only one factor, and neglect to mention his elaborate training, the intimate personal character of his relations with his congregation, and his more absolute authority, as other important elements.

Whatever the shortcomings of the doctor in psychological knowl-

edge may be, the shortcomings of the minister at present are no less, and the latter not only wants the practical experience which serves to guide the physician in these dangerous waters, but may wreck the ship if, instead of the lamp of knowledge, he steers by the unsteady light of emotional sympathy. It seems improbable in such matters as these, involving judgment of character, knowledge of men, and acquaintance with the tortuous workings of the neurotic mind, that a few lectures in a seminary course will enable the average divinity student to do better than his far longer study and more practical contact with men and minds will the medical graduate, assuming both equally equipped in the beginning with the necessary human comprehension and with the gift of sympathy in its best sense.

Nor is it only against overdoses or misapplications of sympathy that the minister who would help the nervous must be cautioned. More serious ills may follow if the use of religious emotion as a motive or stimulant is pushed to excess. It should be remembered that emotional instability is characteristic of many and various nervous disorders, and emotional overstrain among the commoner causes of not a few of them. Care must be used, then, in any appeal to religious feeling, with constant recollection that the force may be a terribly dangerous one to use, and may increase or even create that very overexcitable condition which should be lessened or prevented. Dreadful moral disasters have resulted because a clergyman could not see that religious emotion, a good thing in itself, might prove too strong a medicine for a moral constitution already weakened by emotional excess, or that there were degrees in its applicability and must be graduation in its doses. A thousand times more strongly should this be said of the uses of suggestion as a treatment, since oversuggestibility is in itself a diseased condition, and every use of suggestion (that is, hypnotism), however mild, increases the suggestibility just as cultivation increases the fertility of the soil, so that chance sown seeds of evil suggestion take ready root in ground prepared to receive them, and grow as bad habits are apt to do, even more strongly than good ones.

There is another question raised by the mention a little while ago of the unlicensed practitioner. The man who treats patients by the light of nature we put in jail for the protection of the public. The State holds even the graduate and experienced physician legally responsible, liable to be amerced in damages should he by a mistaken diagnosis or from want of a reasonable sufficiency of knowledge treat his patient wrongly, or confound one disease with another. We have had no suggestion as to how the minister will stand in this respect. Assuredly the time will come when a judicial definition of his responsibility and of his clinical capacity will be demanded.

From what has been said and written it appears that the great field of usefulness of the movement is to be in "functional nervous diseases." The victims of these are to be committed, soul and body, to the clergy. This is the present conservative claim, but certain signs indicate that they will soon take all diseases for their province and omniscience for their foible, as has already happened in the English "Emmanuel movement."

Now what are "functional" nervous diseases? You and I are uncertain, but then we are students and scientific workers. There is no gap in the ministerial knowledge, no hesitation. The authors of *Religion and Medicine* appear to regard any disease as functional that is not accompanied by external evidence of alteration of structure as obvious as a broken bone. This, of course, is not their definition. Indeed, in this matter of diagnosis, as in some others, they avoid definiteness. We know that almost every year new knowledge, closer observation, takes one or two diseases out of the functional list and puts them in the organic class. Moreover, even at the risk of being charged with materialism, one may be permitted to express doubt if there is *any* purely functional disease. Pain in the stomach represents a disturbance of function, but also, according to its cause, hyperemia, anemia, deficient or excessive secretion, etc. The most typical of the functional nervous diseases is neurasthenia, which is also indiscriminately called nervous collapse, breakdown, exhaustion, and, in the forms chiefly affecting the mental side, psychasthenia. We describe this whole class of disorders inclusively as being fatigue neuroses. Is fatigue a functional or an organic symptom? Organic, most certainly. Muscle fatigue is due to poisoning of muscle by waste products of energy. Nervous fatigue is due to changes in the ganglion cells distinctly visible under the microscope. Besides, the diagnostic distinction between organic and functional nervous disease is a convenient rather than a fundamental one. Take again this much discussed prototype of the functional troubles, neurasthenia; many of us think it absolutely due to disturbance of nutrition in the nervous system; that is, in short, to some form of localized tissue starvation dependent upon a great variety of climatic, occupational, and other causes.

The evidence of this is plentiful, clinical and physiological, but too technical for brief statement here. The best wits of the most expert clinicians often find it difficult to decide whether a disorder is functional or organic, even on repeated examination. Still more complicated is the question when the patient is the victim of both functional and organic disease—a combination which may test the acuteness of the best diagnostician and utterly baffle one less well trained and experienced, and might demand not one but a dozen examinations for final judgment.

It is scarcely worth while to waste time on a discussion of the

refinements of diagnosis with an author who says: "All the functional neuroses are to be regarded as diseases of the subconscious mind. By recognizing this we simplify diagnosis."[2] We do, indeed! Diagnosis is a part of medicine just as much as therapy is—and psychotherapy is but one part of general treatment. One main cause of disagreement with the movement I am discussing is the overinsistence upon mental methods to the neglect of physical ones. The possible need of physical treatment is grudgingly admitted, but one might read all their publications and conclude at the end that physical methods were seldom or never required. One author, the Rev. Dr. Batten, in an article on "Psychotherapy," has extracted instances of psychotherapeutic treatment from the Old Testament. Among others, he cites the fact that the witch of Endor gave Saul food, to compose his soul. The witch (even a doctor may read the Bible) had just announced his approaching death to Saul, so that he may have been in need of suggestive therapeutics, but a few verses further on comes the explanation: "There was no strength in him, for he had eaten no bread all the day nor all the night," and the woman begs him to "eat that thou mayest have strength when thou goest on thy way." But one should beg pardon of Dr. Worcester, who, however one may differ with him, must be taken seriously, for speaking of his arguments in the same paragraph with those of Dr. Batten. The latter may be left to the tender mercies of Dr. Joseph Collins, who has handled him and his circular letter with a bitter playfulness which it is to be feared was wasted on a critic whose sense of humor must be as ill developed as his faculty for criticism.

Certain other peculiarities and contradictions should be mentioned before leaving the question of diagnosis, since that involves the selection of cases for treatment. One is that, in spite of much undigested assertion about hysteria and Myers' theory of the subconscious mind, neurasthenia and hysteria are constantly confused. We all know how they run into one another—and how troublesome the borderland cases are—but how can it be asserted by anyone with knowledge that "In almost all such cases (functional neuroses) the real cause of the disorder is moral or psychical?"[3] This would be interesting and valuable, if true, but it is not true. In a vast majority of such cases the real causing cause is physical—and the psychic difficulty is a later addition. It is probably also true that in many of these cases the physical inheritance is bad—not in the sense of inherited disease but in providing a suitable soil for the growth of neurotic troubles and in giving an ill-balanced or oversusceptible nervous system. For a single example of physical cause of neurasthenia take a railroad shock. A man in perfect condition is badly shaken up in a train wreck, with slight external injuries. In two

[2] Religion and Medicine, p. 112.

[3] Ibid., p. 48.

or three days he goes all to pieces nervously and remains so perhaps for many months—to be called weakminded by his friends and accused of simulation by the railroad. It has been frequently proved that if after such a shock the victim is kept quiet, body and mind, for two or three weeks, the bad effects are minimized or entirely prevented. Often a person seriously injured in such an accident escapes altogether the nervous shock—because of the enforced rest required for the treatment of his wounds.

"Psychical disorders give rise to . . . physical disturbances . . . and these may require physical . . . treatment." Many times oftener, probably, the reverse takes place and physical disorders give rise to psychical disturbances. Dr. Weir Mitchell, who is quoted a good deal in *Religion and Medicine,* has advised in *Fat and Blood* and other works on the treatment of nervous diseases, that physical treatment should *precede* moral in order that the latter may be more effectual. It is only in trifling cases of neurasthenia that moral suasion, suggestion, explanation can make a complete cure. When extreme anemia, digestive feebleness, absence of appetite, numberless pains, constant fatigue, asthenopia, and the rest of the endless catalogue of ills and aches are found, the inability of the patient to concentrate attention is often so great, and the mind so distracted and confused as to render it inaccessible to useful or lasting improvement by suggestion. In such conditions suggestion may temporarily cover up the symptoms, but it does not reach the disease. This sort of success is in the eyes of its practitioners the justification for its use, and in those of sober critics a grave argument against it. To cover or to mitigate the more conspicuous and annoying symptoms results in a neglect of the physical states which are at their roots; for example, if, as so good and sane an authority as Dr. Münsterberg asserts, the anemia accompanying neurasthenia is bettered by psychotherapy, it is probably a temporary stimulation in blood distribution that makes this seem to be so, and a careful cellular count would show a deficiency in the blood elements still present, a deficiency which for permanent improvement will need such profound alteration as iron, ample food, and massage will bring about; and so with the other symptoms—the headache, whether due to anemia, to constipation, or to eyestrain, will only be finally better when the causes are removed, not when it is bettered as a glass of wine betters it until the passing effect disappears. In hysteria, on the other hand, miraculous cures may be wrought almost instantaneously by suggestion, no matter what we call it, and thousands of such miracles have been reported by doctors and many more thousands gone unrecorded, partly because these "cures" are prone to relapse.

The experienced neurologist who has seen many cases of nervous break-down in any of its forms not hysterical will agree that cases suitable for purely mental treatment are few and far between. In discussing this subject with Dr. Weir Mitchell last winter,

case records for the previous eighteen months were examined to settle for our own satisfaction how many cases in a large practice would have been suitable for such purely mental therapy. The conclusion was that *three* of our patients might have been so handled with success, but that the result of combined treatment even in these cases was, in all probability, both more rapid in action and more permanent in effect.

The neurologist sees daily patients with organic disease in whom the nervousness has been so conspicuous that the general practitioner has been deceived, stuck on his tag of "neurasthenia," and left it to the neurologist to discover that the patient had structural disease, such as Bright's disease or cancer—or often enough incipient insanity in some form. The delay of a few weeks for the treatment of these diseases by suggestion, by exhortation, by enthusiasm without knowledge, may render impossible their ultimate cure. Is it probable the examination will be more careful, the diagnosis more accurate, when the question is only whether the doctor shall give up the case altogether?

But, of course, the final result is that if there are mistakes they will be the doctor's fault, and if successes, they will be the clergyman's virtue! We Americans have always been like that, and I suppose we shall be until we reach a higher level of civilization. We boast of our education, but it has never gone deep enough to uproot a superstition in favor of amateurs. One has known plenty of educated people who would not consider the opinion of a surgeon if they could get that of a member of a family of hereditary bonesetters. We have insisted in several wars that a saloon keeper would make a better general than a West Point graduate—an opinion which we backed with our best blood. We consume vast quantities of patent medicine, we offer the chief field of the world to quacks, medical, spiritual, and commercial, we hinder every effort at raising professional standards—because we always feel sure that a trained man is never so good at his job as an untrained one.

In Dr. Worcester's latest pronunciamento[4] he forgives his critics beforehand, but has "noted with satisfaction that no radical criticism of our work has proceeded from a man who has studied it at first hand." If by this, as one must suppose from the context, he means that no one is competent to criticise the Emmanuel performances who has not been in actual contact with them, he lays an impossible condition upon his critics, and they might easily ask if this rule would not work both ways and apply equally to the Emmanuel criticism of physicians. How many years' service in hospital must a minister have before he can criticise the practice of medicine? Without comment, it will be enough to set down a few names of those who have adversely criticised the new plan

[4] Century, July, 1909.

and leave to the reader to decide if they are men able to form opinions from the vast and discursive literature of the subject. These publications are presumably meant to enlighten our ignorance, but if we cannot learn enough from them to form views, and must make pilgrimages to the original shrine to see suggestion and hypnotism properly carried out, Dr. Worcester is laying on our shoulders heavy burdens and grievous to be borne.

Conspicuous among these critics, then, stands Dr. Weir Mitchell, who expressed himself upon the subject before the American Neurological Association in 1908. On that occasion Drs. Mills and Dercum, of Philadelphia, agreed with his views, as did Drs. Sachs and Spitzka, of New York, and Putnam, Taylor, and Knapp, of Boston. Of these gentlemen, several have since published detailed and precise criticisms, as have Collins, Hamilton, and Darlington, of New York, Hun, of Albany, and Benedict, of Buffalo—a fair array of names of authority in neurology and in general medicine. Besides these, some psychologists have been unwilling to accept the theories or methods of the clerical psychotherapeutists, and Mr. H. R. Marshall, a former president of the American Psychological Association, Professor Witmer, of the University of Pennsylvania, and Professor Münsterberg, of Harvard, have pronounced against the movement on very various grounds.

To return again to the question of diagnosis, which is interesting, too, in view of the rosy character of the results reported from the churches. They are nearly as completely perfect as the cures at St. Médard. The habit of mind induced by years of observation of cases makes one doubtful of too uniformly successful treatments. One questions if many of the cases must not be of the hysterical type that chase from cure to cure and are always seeking new sensations in treaters and treatment. For these, soothing suggestions and darkened rooms and "gentle pressure of the hand" furnish novel and delightful emotions and fill them with that sense of their own importance so dear to those who "enjoy ill-health."

If one takes up a medical journal, in its melancholy columns he will often find an author recording his failures: he treated, he says, so many patients by such a method, so many recovered, so many failed to improve, so many died. Are there no failures to be recorded under this new plan? What of the ones without faith? The statistics are too good—80 per cent. of cures of alcoholism—indeed! Let us have a definition of alcoholism. If periodical drinking is meant, we can all cure that—between sprees! and the cure will be perfect until the next spree, at which point if one can catch the toper and head him off at the start, he may be stopped (*i. e.*, "cured") again. But what about the enlarged liver, the inflamed kidneys, the atrophied stomach of the alcoholic? Are these cured too? Even taking the broad statement of successes with inebriety or alcoholism, the time since the beginning of the Emmanuel treatment is too short

to be certain of the results remaining satisfactory. The medical profession is rightly suspicious of one of its members whose published statistics are too good. We like to see the failures recorded too. The assertion of the cure of 80 per cent. of alcoholics was made in a lecture in New York, by the Rev. Dr. McComb, assistant rector of Emmanuel Church. At the meeting in Philadelphia, where the paper was read upon which the present article is founded, Dr. McComb first denied and then reasserted this statement, and added that anyone who knew his job ought to be able to "cure" that percentage of "alcoholics." Mere physicians cannot hope to rival the therapeutic efficiency of this gentleman—and since no definition was forthcoming of alcoholism and alcoholics, we must be polite enough to accept his statement on its face; though the description, largely inferential, of the "alcoholism" in cases in *Religion and Medicine* reads for the most part like what might better be described without technicality as "drinking" or even as "hard drinking." Either of these is very different from "alcoholism," as doctors, at any rate, will know. Absent treatment is given to drinkers[5] and reported (by the still absent patient) to be successful. Again, a mere doctor, knowing the morals of drinkers, would want to feel the pulse and perhaps smell the breath of that patient while he is writing his letter.

More serious still appears to me the use of hypnotism in any form or degree. It is perhaps worth while to say that my experience in the practice of hypnotism extends back not two years (the Emmanuel movement began in 1907), but twenty-two. The first case that I can recall in which direct medical use was made of hypnosis was in 1886, in Vienna. After that I worked at it in Vienna and Paris, in 1886, and again in 1890, and have used it occasionally since, but less and less frequently and more and more cautiously as conviction of its dangers grew.

One reads in the official publications of the Emmanuel movement a great deal about hypnotism, in which its use is described and recommended in one place, condemned and denied in another. Suggestion! Suggestion! Suggestion! is the continual word on every page—waking suggestion, suggestion in sleep, autosuggestion, suggestion by another. If suggestion as a medical or medicoreligious measure means anything, it means hypnotism. If not, it had better be called by some other name than by this entirely technical term. If it means only encouragement, persuasion, and reasonable presentation of the case to the patient, or such use as everyone in the world makes every day to bring another person to see and apprehend his point of view, why call it by a technical name? The fact is it does mean hypnotism. Moreover, the descriptions given in *Religion and Medicine*, in Dr. Worcester's lectures, in the letters and literature of a number of the disciples,

[5] Religion and Medicine, p. 54.

speak of hypnotic suggestion as if in constant use. Side by side with these letters and descriptions are found letters denying hypnotism and saying that hypnotism plays no important or frequent part in their treatment.

Dr. Worcester, in a lecture in Philadelphia, described the use of a crystal ball—the oldest and most mechanical method of hypnotization, and said he used it "to fix the attention." To fix the attention for a few moments on a crystal ball, if the eyes are held to it, will hypnotize, to a certain degree, almost anyone, and still more if the other methods described are in use at the same time—quiet surroundings, concentration of mind, and the soothing voice directing attention to the ball and making suggestions. It is unnecessary to labor this point. We may simply say that anyone who says that suggestion in the form which these letters, lectures, and books advise is not hypnotization, does not understand hypnotism.

As to the possible ill effects of hypnotization without bad intent, while numerous instances could be quoted, it may suffice to recall some experiments at the University of Pennsylvania years ago. Two gentlemen, both promising young medical men then in various lines, and one of them now a most distinguished practitioner, experimented upon self-hypnotization or autosuggestion in the production of attacks of convulsion. After a few weeks of occasional experiment at such times as they could spare from other work, they found themselves able to bring on convulsive seizures of a most violent cataleptic description, sometimes with clonic movements. They soon stopped, finding the consequent nervousness and other effects disagreeable, and especially that the convulsions were *beginning to get beyond their control.* One of them told me that he had had convulsive attacks—controllable, to be sure, but sufficiently lively to be disagreeable—from so small an irritation of his nerves as a heavy cart rattling by as he walked in the street would cause. These were not sick men, nor neurotic patients, but strong, active, hard-working, unimaginative young doctors. Plenty of worse cases could be cited, but I will simply repeat the assertion which is backed by almost everyone with large practical medical or psychological experience—that repeated hypnotism decreases the powers of self-control and does *not* increase them, thus most obviously weakening the will; that the surrender of one's personality, one's mental independence, into the hands of another is a serious danger, can be denied by no one who reflects upon the ease with which such mental states become chronic enfeeblement.[6] The quotations in

[6] Mr. Henry Rutgers Marshall (a former president of the American Psychological Association) in a letter to the New York Times, makes the following statement, which needs no reinforcement: Suggestion, he says, is "an effect of one person's act of will upon the other's power of willing. An individual's acquiescence in the reception of radical forms of suggestion is an abrogation by him of his power of willing, and the process by which this suggestion becomes effective is dependent entirely upon the subserviency of the mind of one individual

Religion and Medicine to the contrary, are some of them fitted only to exceptional cases, and some of them are from persons of no recognized authority whatever. Indeed, it is astonishing to find the miscellaneousness with which authors—medical, psychological, and others are quoted in this book, as if the mere names were sufficient to give weight. Bramwell is quoted to offset Janet, for example, or Schofield is cited against Maudsley, as if their views were of equal value. No one could tell from the way they are quoted that Charcot, who touched so many medical subjects and added something of value to almost every one that he did touch, was any better authority than Dubois, whereas every educated physician knows that it would be moderate to say that Charcot's opinion was about six thousand times more valuable than Dubois' on any matter.

I desire to repeat, as the most important contribution to be made to this subject, that the use of hypnotism in any form, manner, or degree is a matter which should be under legal control. It is a dangerous remedy in the hands of the best qualified medical man, and utterly to be condemned when used by any one not a qualified and responsible physician. In Europe our Emmanuel friends would quickly find themselves in the hands of the police, who would probably prove less patient of amateur medical practice than our Boston confrères, who have with scarcely an audible protest consented to the subordinate and undignified position in which the new plan places them.

Nevertheless, I may conclude as I began by saying that in criticising the Emmanuel movement I am finding fault not with the use of proper mental means of cure, but with their improper, unwise, exaggerated, and unscientific application by persons not fitted to judge the needs and limitations of the patients treated. Mental and moral therapy is as good and valuable a remedy as it always was, and although it will suffer discredit, as other good remedies have done, from ignorant enthusiasm, the new attention which the discussion has brought it will in the end do good.

to the mind of another." The one thus guided "agrees to allow his mental states to be determined by outside influences as far as this is possible," and thus the control of the individual's self, so efficient in the building of character, is broken down. He points out, too, that freedom from pain or bad habits or from worry when due to the suggestion of another may be paid for by a weakening of personality. This result is a familiar one to those who have watched the effects of Christian Science treatments and beliefs and observed the calm and cheerful selfishness, the happy readiness with which the convinced and converted "Scientist" shifts his burdens of responsibilities on the nearest person he can find willing to carry them.

THE TREATMENT OF CHRONIC BRONCHITIS.

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To any one who has treated many cases of chronic bronchitis, it is needless to say how unsatisfactory it is. The best we are able to do in most instances is to relieve somewhat; but as to cure, that seems rarely, if ever, to occur—at least among our hospital and dispensary patients. Of course, if we should get, as we sometimes do, a patient who states that during the previous one or more winters he had persistent cough and expectoration, but during the intervals was apparently well, we are hopeful. And this hope is increased, if, after careful inquiry and examination, we find no distinct evidences of heart, lung, or kidney disease, or, indeed, anything abnormal other than what is easily explained by changes of the bronchial mucous membrane.

Now, what should we do, so far as may be, to prevent those conditions from arising as complications or sequels which, sooner or later, would make life miserable and notably shorten it? If the patient be well to do and the sacrifice be possible, we would say, first of all, and as soon as the weather becomes bleak, raw, and changeable, go to a warm and genial climate where life in the open air is agreeable and curative. But few men or women can properly do this when means and obvious duties are considered; at all events, not until it is imperatively necessary. If we wait until this period, secondary changes in different important organs have already taken place, and the best we can do is to keep them stationary, or prevent their getting rapidly worse.

In chronic bronchitis, hygienic measures are of primary importance. Good ventilation, plenty of air and sunshine are essential; so are good food and rest. Clothing suitable to the season, and well selected, is necessary. Apparel too heavy, which overheats in the house and causes perspiration, only brings on fresh colds and aggravates the chronic disease. Food should be simple, well prepared, and nutritious; but heavy meals, especially at dinner, must be interdicted. Strictest moderation in sweets and alcohol should be enjoined; and tobacco, as a rule, hurts notably, except in mild quality and very small quantity in the evening. All the foregoing, and much that could be added about the risks of draughts, wet feet, exposure to cold, biting winds, etc., are among the platitudes.

As to medication: Internally, the iodides are the most useful drugs we have when properly used, and without them our armamentarium in this line would be very defective. For my part my two standbys are iodide of potassium and syrup of hydriodic acid. When there

is dyspnœa, even slight, or nervous irritability shown in any way, we should combine the iodide with Hoffman's anodyne. Hydriodic acid may be alternated advantageously with terpene hydrate in fairly large doses. In the use of the latter drug I agree with my friend, Dr. R. H. Babcock, of Chicago. Invariably counterirritation to the chest should be insisted upon, and kept up for many days, or weeks, with occasional intermissions when the skin becomes tender. Nothing equals the compound tincture of iodine for its resolutive qualities, and the derivative effect toward the skin is all that is desirable. Internally I am opposed to the use of sedatives or anodynes, unless imperatively required. The least objectionable are the combined bromides, henbane, or codeine. A mercurial, followed by Rochelle or Epsom salt, is useful once a week or oftener, and diminishes cough and expectoration for a time in a pronounced degree.

Vapor inhalations, especially of creosote, are very valuable when properly used, and if persisted in are more curative than any other one thing, unless it be change of climate and, at times, habits and occupation. The inhalations should be used with the perforated zinc inhaler; my well-known formula of equal parts of creosote, alcohol, and spirit of chloroform is unequalled. Internally, creosote may also be given with the happiest effects, in small repeated doses, and, combined with the best whiskey and glycerin, will rarely disagree with the patient.

These patients cannot, should not, be housed. If so, they soon get worse, and their bronchial mucous membrane will not bear the slightest change without increased cough and expectoration. If a change of climate may be indulged in, one should go, preferably, to the sand hills of Georgia in winter, and in summer to the Adirondacks, at a moderate elevation. If permanent banishment seems desirable, California, not too near the coast, is the one place of best resort.

In these remarks I have considered the treatment of chronic bronchitis before the advent of emphysema or asthma or dilated heart. When one or the other or all these occur, the indications are somewhat different, more difficult and complicated. When these are not present, moderate exercise in the open air each day should be urged as imperative, unless the weather is very inclement or the patient is suffering from an acute attack grafted on the former trouble. Of all exercise, walking, moderate golf, and riding are the best.

Whenever there is an acute attack, with increased cough, tightening of chest, and less and difficult expectoration, with or without fever, benzoin may be substituted for creosote, and hot water used to vaporize it. In like manner, ipecac, sweet spirit of nitre, and spirit of mindererus take the place of the iodides; and mustard or soap liniment to the chest, and pediluvia at bedtime, take the place of compound tincture of iodine.

It would seem as though one should have more and better facts

to offer. I confess I have none. I have tried many, many things, internally and externally; all sorts of cough mixtures; all sorts of sprays and inhalations, including the globe inhaler, with vaporized oils and divers drugs, and finally, I have adopted what precedes, as best. Of course, judicious tonic medication, with iron, bark, cod-liver oil, arsenic, strychnine, etc., are at times desirable, and undoubtedly indicated. But above all, let it be understood that nothing practically equals dry or moist vapor inhalations of creosote, after the manner I have so frequently affirmed. Indeed, for almost all affections of the respiratory tract, including laryngeal and pulmonary tuberculosis, there is absolutely nothing now known quite or at all equal to them in efficacy and great power for good. But in order to get good, the best, results, one must have practical knowledge how to use them, and faith in their power to help when other things without them fail or are incomplete.

In the way of spa treatment, the only resorts I believe in specially on the eastern side of the United States are Sharon and Richfield Springs, giving the preference to the former, despite the fact that it is not an amusing place. Aix-les-Bains and Homburg, abroad, are the two valuable springs in my judgment. At Aix, one gets Marlios vaporizations; at Homburg, a specially useful innocuous alkaline spring, the Elizabethan, to drink.

AN ANATOMICAL STUDY OF PERICARDITIS.[1]

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Pericarditis is among the most venerable of recognized cardiac lesions in the history of medicine. Galen is said to have observed pericarditis in animals, and to have suspected its existence in man. Among the ancients the "hairy heart" was supposed, according to Haller, to indicate great valor and bravery. Among those heroes who after death were found to be so endowed are said to have been

[1] Read at a meeting of the Pathological Section of the Buffalo Academy of Medicine, April 20, 1909.

Leonidas and Lysander. Morgagni and Vieussens described the obliterative type of pericarditis, and Corvisart, in 1811, noted the significance of the bulging pericardium. Although Laennec was familiar with the lesion, he expressed his doubts as to the possibility of diagnosis, and the characteristic to-and-fro rub of the acute condition was not recognized until 1824 by Collis.

Notwithstanding the antiquity of the anatomical recognition of pericarditis, Osler confesses that it is a condition more frequently recognized in the dead-house than in the ward, referring chiefly to chronic pericarditis. Great difficulty in diagnosis of the acute lesion should no longer exist, unless it occur as a terminal condition in the late stages of some disease when the clinician's attention for detail oftentimes becomes relaxed in the study of obviously fatal cases. In such instances, however, the recognition or its failure is of little practical importance.

During the past three years our attention has been especially drawn to the subject, for the most part, because we have found the lesion so frequent at the autopsy table, and so rarely recognized by us in the wards. Yet the conditions in which pericarditis arises as a frequent complication are in general well understood and fully recognized. We have been constantly on the watch for it in such instances, but nevertheless our percentage of ward diagnoses in cases subsequently demonstrated in the dead-house has been disappointingly small. This study has, therefore, been undertaken not only in order that we might, if possible, correct these errors of diagnosis in the future, but also especially that we might more satisfactorily determine the nature of the process and better estimate the bearing of so marked and striking a lesion, which, however, often exists with very few or no obvious clinical signs.

The cases on which this study is based have been for the greater part taken from our own personal service; a few are from the routine protocols of the pathological department at the University and Bellevue Hospital Medical College. Our clinical observations have been chiefly made in the service of one of us at the City Hospital. In all, 1000 protocols have been examined, and of these, 150 have shown pericarditis.

Only frank and outspoken pericardial lesions of true inflammatory nature have been considered in this study. In the acute cases, for example, no instances of hydropericardium have been included, and we wish to make a sharp distinction between pericarditis, a true inflammatory lesion, and the hydropericardium, which occurs in various œdematous conditions. This may, indeed, be followed by true inflammation, but it is not in itself primarily of this nature. Of the chronic, only such instances have been included as showed manifest inflammatory thickening of the membrane or adhesions of considerable extent. The small granulomatous patches which are found so frequently, especially over the auricles and cardiac base,

for example, have not been included in these statistics, although these changes are also, in our opinion, unquestionably inflammatory.

Classification and Pathological Anatomy. For clinical purposes pericarditis is best classified as acute, subacute, and chronic. Subacute or chronic pericarditis occurs in many instances as an extension or terminal state of the acute forms in which there has been a fibrous or granulomatous replacement of the acute exudate.

In both acute and chronic forms the process is usually found most marked on the visceral layer of the membrane, even in those cases in which the lesion has been produced by direct extension, as from a pleural, pulmonary, or mediastinal inflammation. The most logical explanation of this fact appears to be the greater physical activity and the greater vascularity of the epicardium, as compared to the parietal layer of the membrane.

The acute forms are subclassified as fibrinous, serofibrinous, hemorrhagic, and purulent. We have found the division into fibrinous and serofibrinous unsatisfactory, since in both instances the lesions are practically identical. In the one, more clear serum is exuded; in the other, this serum is richer in coagulated fibrin; we shall, therefore, in this study group together all cases ordinarily classed separately. Similarly, hemorrhagic pericarditis is almost without exception the simple adding of extravasated blood cells to the serofibrinous exudate or to a purulent one, and we have, therefore, for statistical purposes considered hemorrhagic cases under the heading of one or the other of these forms. By some authors, however, hemorrhagic pericarditis has been considered as diagnostically definite; thus, Thorel states that it is present in tuberculous tumors of the pericardium, in uremia, the various hemorrhagic diatheses, and in leukemia. We have found it to have even a broader etiological basis and to be shown in practically any severe type of pericardial inflammation. Stewart,[2] for example, reports it present in a case of infection of the pericardium by the colon bacillus.

Acute serofibrinous pericarditis is a lesion characterized by the primary appearance of minute granulomas on the surface of the pericardium. The capillaries are injected and proliferation of the endothelial and connective-tissue cells is set up, together with inflammatory infiltration of the entire depth of the membrane. The change is seen most frequently or most marked over the base of the heart and notably over the walls of the auricles. Only the epicardial layer may be primarily involved, but sooner or later a similar change appears in the adjacent parts of the parietal layer. At the outset the lesion is very apt to possess a patchy character, but eventually in progressive instances it finally becomes diffuse. Coincident with the inflammatory changes mentioned a deposit, first, as a thin pellicle of fibrin gathers over the eroded surfaces, and this may increase in

[2] Zentralbl. f. innere Med., 1904, xxv, 863.

thickness, varying in different instances from a very delicate web to a coat as much as 1 cm. in thickness. Serum and leukocytes exude into the sac from the distended capillaries, and the amount which may so collect may become very great, as much as 2 to 3 liters, according to Osler and Gosselin, while Kestner claims to have found from 3 to 10 liters in scorbutic cases—all amounts far in excess of the fluid which can be artificially injected into the normal sac post-mortem, as shown by the experiments of Smith, Lusk, and others. Minute petechial hemorrhages appear in a good many instances, either into the fibrinous deposit or more frequently into the sub-endothelial connective-tissue layer, and in some cases actual granulation tissue may appear. Of course, when the pericarditis develops in such conditions as the hemorrhagic diathesis, in purpura or scurvy, the hemorrhagic exudate is very pronounced. Hemorrhage is also frequent in tuberculous inflammation and in those inflammations which arise from neoplastic invasion of the pericardium. In these last-mentioned circumstances blood extravasation takes place directly into the free fluid, and is not limited to the substance of the membrane or its adherent exudate.

It is generally admitted that in the healing of serofibrinous pericarditis fibrous substitution of the exudate takes place, and when abraded surfaces are apposed, adhesion, of course, with resulting synechia at these points follows.

The bacteria found in serofibrinous pericarditis commonly bear a direct etiological relation to the process. Those most commonly present are the streptococcus, the pneumococcus, various staphylococci, and, according to Poynton and Paine, Diplococcus rheumaticus. Secondary infecting organisms of all sorts may, of course, be present, and in some cases the exudate is sterile.

Purulent pericarditis in our experience occurs either from direct purulent infection of the pericardial sac or as a sequence to the serofibrinous form when infection with pyogenic organisms takes place. In most instances the effusion is less abundant than in the cases of serofibrinous inflammation, but it may become great before death ensues. As a rule, especially when the condition follows the serofibrinous form, the exudate is rich in fibrin, although the shreds are apt to become more or less liquefied. Pus, eroded connective tissue and endothelial cells, and general inflammatory detritus are also abundantly present in the fluid. The portions of the membrane previously most deeply covered by fibrin are now commonly overlaid by granulations. The color of the purulent effusion is, of course, chiefly dependent on the nature of the infecting organisms. Gas may also appear due to the action of microbic growth. Whenever the case has been of long standing one is very apt, in addition to whatever organisms may have been first present to find the colon bacillus or members of the proteus group. We have twice found Bacillus aërogenes capsulatus in purulent pericardial effusion when general

infection with this organism was present; gas was, of course, abundant in both these cases. The tubercle bacillus, streptococci, and staphylococci are the most common organisms found, and they usually bear a direct etiological relation to the disease.

When healing follows purulent pericarditis, extensive fibrous adhesions almost invariably form, and in some cases we have found patches of calcification.

Chronic pericarditis is most conveniently divided into a chronic fibrous variety, in which adhesions either do not exist or are present to very limited extent, and those cases which show chronic fibrosis with adhesions. The latter is by far the most frequent form in our experience. In the simple chronic fibrous form the essential process is a thickening of the fibrous layers of the membrane due in many instances to inflammatory changes, but frequently to a general fibrosis with this local manifestation, as in certain types of syphilis, akromegalia, arteriocapillary fibrosis, and the like. Hess[3] thus describes this lesion as a local manifestation of a general chronic serositis. The thickening may be entirely limited to a single layer of the pericardium, in which instance it is almost invariably the visceral layer which is so involved, but in most examples, and especially when it follows a previous serofibrinous or purulent inflammation both layers are diseased, but the visceral to the greater degree. Occasionally one finds extensive plaques of thickening without general involvement of the sheet, and here the changes are most prominent about the base and over the auricles.

Microscopically, the alterations are briefly those of a fibrous hyperplasia, usually with more or less still active round-cell infiltration, and commonly with exudation of some extent. In this variety, and also in cases of fibrous pericarditis with adhesions, when the etiology is tuberculosis or syphilis, the characteristic lesions of these infections may be present, although by no means invariably.

In chronic pericarditis, with adhesions, a very wide range exists between those cases in which only a few fibrous strands unite the visceral and parietal membrane and those in which the union is complete and of dense scar tissue. In our statistical studies we have placed in this class only those cases which showed fairly extensive synechia, but it is, of course, manifest that all degrees of adhesion may exist, depending probably on the nature and extent of the primary process and on its rapidity of absorption or replacement. In our experience when the union has been incomplete we have found the most numerous adhesions immediately about the apex or just over the auricles, but in this particular the cases vary greatly.

Tuberculous Pericarditis. As has been stated by Osler, pericarditis caused by tuberculosis may manifest itself either with distinct and demonstrably tuberculous lesions, as miliary tubercles or caseous

[3] Eine Klinische-experimentelle Studie, Marburg, 1902.

masses, or it may be found as a simple fibrous thickening of the membrane with or without adhesions. Although in the latter case the change is unquestionably indirectly due to the tubercle bacillus, if the specific bacilli or distinctly tuberculous changes cannot be demonstrated, we have chosen to class such examples as chronic fibrous or adhesive pericarditis, due to tuberculosis and not as true tuberculous pericarditis. In most instances of this nature it has been found that the pericardial change is but a local example of a more or less general fibrosis. As will be shown later in our study, tuberculous infection of the pericardium may cause practically any sort of anatomical pericarditis, from serofibrinous or hemorrhagic to purulent, fibrous, or adhesive, a fact which we think should be more generally recognized. This may depend on the varying degree of virulence of the organism, on the resistance of the body and its reaction, but probably still more on the nature of the contaminating organisms.

In tuberculous pericarditis of the chronic variety the membrane is, in our experience, generally much thickened and is studded with yellow necrotic areas, which are softening tubercles. When, on the other hand, the change is but a chronic stage of an acute tuberculosis of the serosa, as in some slow cases of general miliary tuberculosis, obliteration of the cavity may not be present, but only occasional shreds of adhesion are demonstrable. In practically all cases of tuberculosis of the pericardium, tubercle formation is not diffusely present, but is associated with inflammatory changes of clearly simple nature, and organisms other than the tubercle bacillus are also etiologically concerned. Indeed, in most cases, from a study of the lesions one is impressed with the idea that the tubercle bacillus is here a relatively inactive organism, and that other agents are probably much more directly involved in the process.

Syphilitic Pericarditis. Practically the same conditions obtain as in tuberculosis. Unless distinctly syphilitic lesions can be demonstrated in the pericardium, an admittedly rare condition, the lesion should not be classed as syphilitic pericarditis, although, as we show later on, various forms of pericarditis may be caused by syphilitic infection, a fact not generally recognized. There is now no doubt that any of the various anatomical forms of pericarditis, either acute or chronic, may be caused by the syphilitic virus. As might be expected, however, only a relatively small proportion show typical syphilitic pictures. In these, accompanying syphilitic changes in the myocardium are the rule. In passing, however, it is interesting to note that in the two instances of gumma of the heart, which occurred in the service of one of us, no pericardial changes of note were present. In the average case of syphilitic pericarditis the membrane shows a more or less diffuse thickening, and it is only on microscopic examination that changes suggestive of syphilis appear. It is manifested usually by perivascular infiltration, nuclear fragmentation, and a distinct tendency to necrosis, as well as toward hyperplasia of the

connective tissue. It is probably partly for this reason that syphilitic pericarditis has been so rarely reported. Gumma of the pericardium has never occurred in our experience, and McPhedran states that but three cases have been recorded.

Traumatic pericarditis does not deserve a separate classification from the pathological standpoint, since in most instances the actual pericarditis is due not to the traumatism, but to infecting agents introduced with it. It occurs mostly in surgical practice, and the changes induced depend on the nature of the contributory infection. The cases of perforation of the pericardial sac with foreign bodies from the œsophagus and stomach are very interesting, and the consequent pericarditis is invariably of a purulent type. The elder Flint thus cites in his text-book a case following perforation and infection from the swallowing of false teeth. Personally, we have never seen a case of this nature in man, but it is common in the ruminants, and several years ago one of us reported such a case in a buffalo, when the perforating body was a bale wire, quite a common circumstance, I am told, among domestic animals.

Mechanical pericarditis also does not exist as a distinct pathological entity, although it is a clinical condition of some importance. It is seen most frequently as a serofibrinous inflammation, occurring in such conditions as dilatation of the heart or from overaction, oftentimes with more or less dilatation. It is seen, for example, in Graves' disease, two marked instances of which have been observed in our service. Although the most common anatomical change found in these cases is a serofibrinous inflammation, fibrous hyperplasia also occurs with relative frequency. Rubino produced acute pericarditis in animals by experimental trauma; and we all have noted its frequency in tube-fed animals.

Knox[4] describes a further form of pericarditis characterized by the appearance of small fibrous nodules along the course of the arteries of the epicardial layer, not entirely unlike the syphilitic form described by Balzer.[5] According to Knox this pericardial inflammation is caused primarily by lesions of a degenerative character in the walls of the coronary arteries. We have not personally observed this form, except, perhaps, in those cases due to marked coronary arteriosclerosis, when the superjacent epicardium was thickened, hyperplastic, and more or less infiltrated; we have not considered such instances as frank pericarditis in this study.

Etiology. It is very questionable if a primary idiopathic pericarditis exists. At least, if it ever occurs it is a very rare condition, and even in reported instances Thorel[6] discusses the probability of most of the recorded cases being tuberculous or the result of hemic infections of other varieties. It is certain then that pericarditis is, almost with-

[4] Jour. Exp. Med., 1899, iv, 245.
[5] Archiv der Phys., 1883, vi, 93.
[6] Ergeb. der allg. Path. u. Path. Anat., 1909, S. 921.

out exception, a secondary lesion or a complication in some other disease or dyscrasia. This statement is now generally accepted, and holds true except for the relatively unimportant traumatic cases. In general our study upholds the usual conceptions in regard to the etiological factors chiefly concerned in the production of pericarditis, but in the investigation of the literature of the subject we have been impressed with the fact that in most reports the statistical conclusions are based on clinical findings only, or in some instances on bacteriological investigation alone, and are, therefore, less apt to be accurately based than when the data have been derived from complete postmortem examinations aided in many cases by histories, clinical notes, and bacterial examinations, as in our statistics.

In the acute serofibrinous type of pericarditis lobar pneumonia leads as the most frequent cause, and was present in 26 out of the total of 67 instances. This is in direct contradiction to the statement of von Schrötter, who states that pneumonia is not shown in his observation as an especially frequent cause of pericarditis. This apparent discrepancy is largely explained by the clinical observation, which I am sure has been apparent to all of us, namely, that certain epidemics of pneumonia have been characterized by the frequency of pericarditis as a complication, and in other years other complications have been more common. For example, nearly all the hospital physicians in New York City remarked on the great frequency of pericarditis in the pneumonia epidemic of 1906, during which time my statistics were partly collected.

In most instances pericardial infection in lobar pneumonia has taken place by transmission by direct contact in so far as could be determined by the local condition of the adjacent pleura and mediastinal tissues, although, of course, lymphatic or hemic transmission cannot be excluded in this general infection. Four cases of pericarditis of the serofibrinous type were found to have developed in the course of bronchopneumonia, and three from simple pleurisy without pulmonary involvement.

Rheumatism has been quite generally reported as the most frequent condition in which pericarditis of the serofibrinous variety develops. When we recall that under the head of rheumatism must be included many presumably septic conditions incorrectly diagnosticated, it is readily understood why this disease has been so generally accepted as the most frequent cause of pericarditis. In our observations, which it must be remembered are founded on postmortem and not on clinical evidence alone, rheumatism was found to be the factor in but 7 out of the 67 instances. Von Schrötter reports acute rheumatism as the cause in 30 per cent. of cases, von Harrass in 7 per cent., Pribram in 5.2 per cent., and Eichorst in 3 per cent. We must, however, not fail to recollect that in this respect clinical data may sometimes be the more valuable, since the larger percentage of these instances of rheumatic pericarditis recover, leaving no traces which

can be afterward definitely ascribed to rheumatism anatomically. Again, in regard to rheumatism all clinical observers are united in admitting that the percentage of pericarditis as a complication varies greatly in different years. Poynton, in his book, states that he has isolated Diplococcus rheumaticus from the pericardial effusion in a high percentage of cases of pericarditis, and he, therefore, assumes that all these instances are examples of true rheumatic infection. Until the relationship between acute rheumatic fever and the diplococcus of Poynton and Paine has been more definitely demonstrated most of us will be inclined to accept this evidence as far from positive.

Six of our cases developed in the course of general sepsis and two took place in generalized tuberculosis, without, however, the presence of tubercle bacilli or tubercles in the pericardium; in other words, they were the result of the mixed infection.

But five instances of serofibrinous pericarditis developed in the course of renal disease, a considerably smaller percentage than is shown in most statistics, and probably accounted for by the fact that our data were collected from postmortem and not clinical records. Four were due to acute and one to chronic nephritis.

Five cases developed as a result of syphilitic infection, four in the tertiary stage. In these examples no typically syphilitic lesions were demonstrable in the pericardium, although the predisposing influence was clearly syphilitic infection. Alcoholism, acute myocarditis, and asthenia appear as the etiological or determining factors in two instances each. In but one case could no probable etiology be found, but this instance was not submitted to bacteriological investigation.

Finally, in regard to the etiological factors concerned in the production of acute serofibrinous pericarditis, it will be observed that general bacterial infections predominated in a high percentage (46 out of 67).

In our eighteen instances of purulent pericarditis generally septic conditions naturally lead, although this was demonstrable in but seven. Lobar pneumonia with apparently direct transmission of the infection was present in three instances and purulent pleurisy in two. Three formed the terminal picture, following rupture of a tuberculous abscess of the lung with direct invasion of the pericardium, but without the formation of tubercles, although doubtless these would have developed had the patient not promptly died from the resulting general septic condition, of which the pericarditis was evidently but a part. Three cases of apparently idiopathic purulent pericarditis appear in which, while a generally septic condition was present, no other presumably primary focus of suppuration could be discovered.

But eight instances of chronic fibroid pericarditis independent of adhesions occur in our series. Of these, tuberculosis (without tuberculous lesions in the pericardium) leads in four examples, and chronic nephritis appears as the probable exciting cause in two.

In chronic adhesive pericarditis, sixty-one instances of which occur

in our list, tuberculosis, which was demonstrable in 17 cases, appears as the most constant etiological factor. In none of these were tuberculous lesions present in the pericardium, and the change seems then to have been induced by the general toxemia of the disease rather than by the immediate action of the tubercle bacillus. This statement is in general accord with the conclusions of most of those who have made a special study of pericarditis in its relationship to tuberculosis. In most instances the fibrosis does not appear to have been the result of a direct transmission of the inflammatory process from the lung and pluera, but to have followed the toxemia of the disease. Osler, for example, states that as a cause of pericarditis tuberculosis follows hard upon acute rheumatic fever.

Arteriocapillary fibrosis, with the pericardial lesion as a local manifestation, appears as the second most frequent cause of this form of pericarditis. It was present in 11 of our cases. We believe that the importance of this etiological factor has been very commonly overlooked by most writers, who have in many instances attributed the changes to nephritic disease, the "uremic pericarditis" of Banti, whereas the renal changes were but a part of the end result of the primary arteriocapillary disease. Independent and clearly etiologically important, nephritis could be traced in but 2 of our cases, while Bulil finds it causative in 35 per cent. of his, Mamberger in 14 per cent., and Frerichs found it probably causative in 18 out of 292. Banti,[7] in two carefully observed cases, came to the conclusion that the nephritic type of pericarditis is caused by the toxic factors present in nephritis, and states that he was able to reproduce the lesion experimentally. It seems highly probable that the frequency of terminal infection in uremia may bear some relationship to the development of pericarditis in nephritic conditions; although perhaps the distant cause of the lesion, it would not be so considered in our study.

Syphilis, with its general tendency to fibrosis and inflammatory changes, has been found as the most probable factor in 10 out of our 61 cases. In none of these, however, could definitely syphilitic alterations be demonstrated in the pericardium, and we, therefore, assume that the pericardial lesions were but local evidences of the general tendencies shown in this disease. We believe that the relationship of syphilis in this respect has been very generally unrecognized by previous writers on this subject, most of whom exclude syphilis as the probable cause unless definitely syphilitic changes are demonstrable in the membrane. This is by no means in accord with the accepted bearing of syphilis on such conditions as hepatitis, nephritis, and myocarditis, all lesions very similar in nature histologically to chronic adhesive pericarditis. Alcholism, with an anatomical picture somewhat similar to the changes found in cases of syphilitic

[7] Ueber urämische Pericarditis, Zentralbl. f. path Anat., 1894, 461.

nature, appears in nine instances out of the sixty-one, and we find in going over the literature that the relationship of this chronic drug poisoning has been also very generally overlooked by most authors, many of whom do not even mention it. Even a slight knowledge of the nature of the general changes which occur in chronic alcoholism is such as fully to convince one of its probable important bearing on pericarditis, particularly of this form.

Chronic endocarditis was concerned in but four examples of pericardial adhesions, in all of which the mechanical factors resulting from irregular action and cardiac dilatation appear to have been prominent.

The relatively small occurrence of rheumatism as an etiological factor in these chronic cases is notable in our series. It was found in only four cases. This probably is misleading, for again our statistics have been mostly founded on anatomical studies, and in some instances the histories were either entirely wanting or deficient in regard to this important question. Further, other chronic conditions are very apt to be associated with rheumatism to such a degree as to distract one's attention from the rheumatic factor; thus, alcoholism, tuberculosis, syphilis, and arteriocapillary fibrosis are all conditions frequently following or associated with rheumatism, any of which present more characteristic and definite anatomical changes. Personally, we are fully convinced that the general opinion in regard to the close relationship especially between rheumatism and the chronic adhesive form of pericarditis is correct.

But 6 cases of true tuberculous pericarditis were found in our series of 150. None of these were primary, and, indeed, primary tuberculosis of the pericardium is a most rare condition in the literature; that is, cases in which actual tubercle bacilli or tubercles have been demonstrable in this membrane and not elsewhere. All of our six instances developed in the course of a more or less generalized tuberculosis, that is, in tuberculous bacteremia rather than in merely localized tuberculosis of the lung or pleura. This fact is especially interesting when we recollect that up to recently it was rather generally conceded that a high percentage of cases of pericarditis with adhesions were really tuberculous. This false impression seems to have originated from the great frequency of tuberculous toxemia as a cause of chronic pericarditis, and because of inadequate anatomical study of the thickened membrane in cases of pericarditis with adhesions. Considering the great frequency of tuberculosis in its relationship to pericarditis, the relative immunity which the pericardium enjoys against this type of inflammation is, indeed, striking.

A very similar condition of affairs exists in regard to syphilis in its relationship to pericarditis, and the relative immunity here, as in tuberculosis, can perhaps be best explained by the theory of McPhedran, who asusmes that it is due to the relatively slight vascularity of the membrane. In but a single case of our 150 could definite acute

syphilitic changes be shown. This was an example of early but very severe secondary syphilis, in which death followed from perforation of an acute syphilitic ulcer of the aorta, but in which an acute syphilitic granulomatous pericarditis with inflammation of nearly all the serous membranes was demonstrable. We have already discussed, however, the important relationship which syphilis probably bears to the development of the chronic adhesive form of pericarditis. Rosenthal,[8] however, states that although it is generally assumed that syphilitic pericarditis occurs only in conjunction with syphilitic disease of the myocardium, it may do so independently, and Schönemann also reports such an instance. The cases recorded by Virchow, Ricord, Mracek, and Herxheimer all showed definitely syphilitic involvement of the myocardium.

No discussion of the occurrence of pericarditis in diseases of infancy, as in measles, scarlatina, and the like, has been presented, as our experience has been limited to adults.

Although there is more or less general unanimity of opinion in regard to the conditions in which pericarditis is prone to arise, the immediate local determining factors which in any given case cause the onset of the pericarditis are by no means clearly shown. It has already been pointed out that on account of the relatively small blood supply of the membrane it is rather less likely to become involved in bacteremic processes than other similar membranes, as those of the brain, for example. In this regard the observations of Charrin, however, tend to demonstrate that the metabolic products of bacterial growth when in the proximity of the pericardium tend to favor involvement of the membrane.

The predisposing influence of local traumatism, even though of slight degree, has been apparently fully demonstrated, the principle involved being apparently that of the locus minoris resistentiæ. This well-recognized fact has been corroborated by the experiments of Banti, who first caused irritation of the membrane by the injection of turpentine or by the mere puncture with a platinum needle, subsequently producing an experimental bacteremia with the pneumococcus, when a pneumococcic pericarditis was quite constantly produced. Rubino caused a typical pericarditis by the preliminary infliction of a blow over the pericardium or even over the thorax at large, and the subsequent injection into the blood stream of virulent staphylococci. Somewhat similar experiments have been conducted with the diplococcus rheumaticus by Poynton and Paine. Stern[9] thinks that other and less definite injuries to the thorax may be the determining factor, especially when mediastinal hemorrhages are induced. These observations are in a way corroborated by certain experiments of our own, in which we have noted the great fre-

[8] Berl. klin. Woch., 1900, Nos. 47, 48.

[9] Ueber traumatische Enstehung innere Krankheiten, Jena, 1900.

quency of pericarditis, especially of the serofibrinous form, in animals tube-fed for considerable periods, in which traumatisms to the mediastinal tissues resulted from the frequent passage. Pericarditis is very prone to develop in these animals even when no bacteremia preëxists, although in most of our experiments the general resistance of the tissues was already reduced by drug administration and terminal infection was, of course, thereby favored.

Clinical observation has also led us to consider the role which overaction of the heart plays, as in Graves' disease, in nephritis with elevated blood pressure, and in the numerous conditions in which rapid action of the heart oftentimes with more or less dilatation of its chambers occurs. Although as yet our data do not warrant us in more than a very tentative statement, we believe that these factors do play a very important role in the determination of pericarditis in cases in which it might not otherwise develop.

The Association of Other Lesions with Pericarditis. In this relationship more interest is derived from the bearing of the concomitant lesions upon the pericarditis than of the pericarditis upon them, for while it may be caused by or follow in the wake of several other lesions, there are very few lesions which follow or result primarily from pericarditis. Nevertheless the association of concomitant lesions in the heart and other viscera with pericarditis becomes at times of diagnostic and prognostic value, more so, however, as indicative of the general process or condition in which the pericarditis has originated than of any direct value in the immediate diagnosis or management of the pericardial change itself.

Entirely as one would expect, the lesions most constantly associated with pericarditis are changes in the pleura. In our 150 cases pleural lesions were present in 136. In well over half of these cases it appears that the pleural change is the more anterior, and that the pericardial alteration occurred later, perhaps even as a result of the pleural lesion, though still back of this relationship is the general disease or condition in the course of which the two membranous inflammations developed.

A direct relationship between pericardial and pleural lesions is best shown in the acute serofibrinous cases, in which out of the total of 67 instances, 60 showed pleural changes; in 36 of these the lesions were identical in character. In 11 others chronic inflammatory changes were present in the pleural sheet, which, nevertheless, also appears to have had a direct bearing on the onset of the adjacent pericardial inflammation. Four cases of purulent pleurisy appear to have been the point of origin of the acute serofibrinous pericarditis, so that in 51 out of 67 cases the pericarditis seems to have appeared as secondary to a like or similar process in the pleura.

This pleuropericardial relationship is even more clearly shown in the cases of purulent pericarditis when associated changes were present in all except one. Purulent pleurisy was present in 9 out of the

18 cases, and in 5 others the pleural change was serofibrinous in nature; possibly, however, in these last five, secondary to the pericarditis. In 3 out of the 4 remaining cases the pleural changes were chronic inflammatory.

The association between chronic adhesive pericarditis and pleurisy is less definite, but the pleura was found involved in 48 out of the 61 cases. In 31 of these the pleural change was like that in the pericardium, which would seem to suggest a common origin. It is very important to note in this regard that in but two instances was the pleural inflammation demonstrably tuberculous when the pericardial change was simply chronic inflammatory in nature. In three examples the pericardial lesion certainly was not secondary, but perhaps rather causative of the pleural change, since the pericarditis was a chronic adhesive one and the pleurisy acute. In six instances both acute and chronic alterations were demonstrable in the pleura.

In our 6 cases of tuberculous pericarditis, tuberculous pleurisy was present in 4, and the other 2 were examples of general miliary tuberculosis, in which for some reason the pleural membranes appear to have escaped invasion.

Although some authors have assumed a more or less direct relationship between pericarditis and endocardial disease, this does not seem to be borne out by the study of our series. From our records it seems even highly problematical if there be any significant relationship between these processes. Thus, of our serofibrinous cases, 46 of which were caused by bacteremia of some variety, only 10 showed acute endocardial changes; 7 of these were unquestionably rheumatic, and bore no other relation to the pericarditis than that both had a probably common etiology. The fact that 28 out of these 67 instances showed chronic endocardial changes is believed to be of no bearing, but to be explained by the great relative frequency of chronic endocarditis. All these cases were very carefully gone into under the supposition that deficient muscular action, with perhaps dilatation and mechanical friction might have predisposed toward the pericarditis. This probability could not be sustained in any of these instances except when other changes to be mentioned later on were present.

This lack of correlation between endocardial and pericardial disease is further emphasized in the purulent cases, which although, universally due to bacteremic conditions were unassociated with endocardial lesions of an acute character, except in one instance. From these cases one may apparently more or less definitely assume that infection is rarely borne by the route of the cardiac vessels from the endocardium to the pericardium, or vice versa.

Acute endocardial alterations were entirely absent in the 61 cases of chronic adhesive pericarditis, but in 35 chronic endocardial inflammation was present, but in no such degree or form in any as to indicate probable dependent or etiological relationship.

Myocardial alterations are present in the majority of cases of pericarditis, but that this lesion has more than an accidental or common etiological relation in most instances does not seem likely. Nineteen of the acute serofibrinous cases were accompanied by acute parenchymatous degeneration of the heart muscle. Study of these shows that in but a very small number was the pericardial lesion in any likelihood responsible for the myocardial disease, which in most cases was clearly due to the general toxemia of the disease in which the pericarditis had developed. On the other hand, a certain number of cases are present in which the dilatation and deficient muscular action of the myocardium consequent upon the myocardial disease may have had a predisposing or determining effect in the evolution of the pericarditis. This suggestion is given further probability by the occurrence of eighteen examples of fatty degeneration of the myocardium in which no derivative effect of the pericarditis can be assumed, but rather the contrary, and this, in our belief, is one of the factors which favors the appearance of terminal pericarditis in the numerous conditions in which myocardial degeneration or deficiency is shown. That fatty degeneration bears an especially important relationship is also suggested by the fact that but six instances of fibroid myocarditis were associated with the pericarditis, a very sharp contrast to the occurrence of the fatty degeneration, which in most cases was further suggestive by the concomitant occurrence of more or less dilatation. In 20 of the acute serofibrinous cases absolutely no myocardial lesions could be made out, and it is most striking to note that only 2 of the 67 showed acute inflammation of the myocardium.

This lack of effect on the heart muscle of the adjacent inflammatory process is still more vividly illustrated in the purulent cases, of which only two showed acute inflammatory lesions in the muscle. Nine of the 18 naturally showed parenchymatous degeneration of greater or less degree, and in 6 fatty degeneration was associated.

All of the 8 cases of chronic fibroid pericarditis without adhesions showed myocardial lesions, among which there were 2 of fatty degeneration, 3 of fibrosis, 2 of brown atrophy, and 1 of an acute and incidental parenchymatous degeneration.

Of the 61 cases of chronic pericarditis with adhesions, myocardial changes were entirely absent in 13. Fifteen showed fatty degeneration, 14 fibrosis, and 7 brown atrophy. Careful study of these cases in particular was made in order that, if possible, some conclusions might be drawn as to the effect on the myocardium of pericardial adhesions. In so far as we can conclude in those instances in which extensive effects on the heart were associated with pericardial adhesions, the myocardial disease was in most cases clearly the earlier, and we fail to find in our series of cases what we have also failed to demonstrate clinically, namely, that adhesive pericarditis has any very important or serious effect on the anatomical condition of, or on

the action of, the heart. In so far as we have been able to observe, some of the cases showing most pronounced adhesions clinically manifested little or no evidence of cardiac embarrassment *unless the myocardium itself was diseased,* usually primarily or independently so.

None of our six cases of true tuberculous pericarditis showed tuberculous invasion of the myocardium, and in none were the myocardial lesions apparently due to the pericarditis.

Renal changes are so universally present in all cadavers that it is very difficult to make an intelligible analysis of the occurrence of changes in these organs in association with pericarditis. Manifestly, in those cases showing arteriocapillary fibrosis alterations of this character were present in the kidneys, but in no instance were we able to find anything indicative of any inter-relationship between pericarditis and renal disease. Neither do we find from our study any indication that renal disease per se is an important factor in the causation of pericarditis, except in a very general or indirect way, for example, when, as suggested by Thorel, pericarditis arises as a terminal infection or perhaps follows myocardial disease of renal origin.

The Cause of Death. One of the most interesting parts of our study has been the investigation of the cause of death in pericarditis. This, we find, is only very rarely due to the pericarditis itself. In but 6 of our total of 150 cases was pericarditis mentioned as among the causes of death. In five of these instances the lesion was septic in nature, and in the one remaining, serofibrinous, due to a general infection, with its most pronounced focus in the pericardium. In most cases the causa mortis has been the general disease or condition in which the pericarditis had arisen as a complication. It is also interesting to note that notwithstanding the admitted frequency with which rheumatism acts as the cause of pericarditis, death was found to result from definitely rheumatic changes in but two instances, both examples of serofibrinous pericarditis.

Naturally, septic conditions are found to be the most frequent cause of death in pericarditis, and in 86 of our 150 cases death resulted from infections exclusive of tuberculosis and syphilis. In some of these, as in 17 cases of chronic adhesive pericarditis, the infections were apparently terminal in character; 8 of them were thus pneumonic and 9 simple sepsis. In the serofibrinous cases the infections were for the most part not only the cause of death, but also the obvious cause of the pericarditis. In the serofibrinous cases it is interesting to note that the cause of death was an inflammatory process adjacent to the pericardium in 30 instances of lobar pneumonia, 4 cases of bronchopneumonia, 1 case of empyema, and 9 cases of acute endocarditis. Not included in this list are four instances of death from acute myocarditis and four from acute tuberculosis.

In chronic adhesive pericarditis the cause of death appears to be absolutely independent of the pericardial lesion, which we have come

to consider as of very little prognostic importance. Pericarditis was not even mentioned as among the causes of termination in any of these cases, and in but 5 instances was a cardiac dilatation the immediate cause of exitus. After a careful study of these cases, in three only can the pericardial inflammation or adhesions be considered as probably actively concerned in the cause of death. Chronic endocarditis was the terminal factor in 4 instances, acute endocarditis in 1, lobar pneumonia in 8, sepsis in 9, empyema in 1, and tuberculosis in 9. General changes attributable to alcoholism appear as the probable cause of death in 6 instances, and definitely syphilitic lesions in 4, myocarditis of non-syphilitic nature in 4, and traumatism in 2.

In the chronic fibrous form of pericarditis without adhesions the cause of death is even more indefinitely connected with the pericardial lesion, and, taken as a whole, we again wish to express our conviction of the very minor role which pericarditis in any form plays in prognosis, except as a measure of the degree or extent of any general infection.

CONCLUSIONS. In conclusion, we wish to summarize those points which have especially impressed themselves upon us in the course of this study, although some of the evidence leading us to these conclusions has been but incompletely expressed in the paper:

Pericarditis is a lesion secondary in nature, rarely or never primary. The complication, for such it must be considered, is in most cases not in itself serious except when of the suppurative variety, and in all instances it is more noteworthy as indicating the general condition in which it arises than on account of its own importance. It is, therefore, of little relative clinical importance, except as a diagnostic index, and in suppurative cases. True myocarditis is infrequently associated with pericarditis, but myocardial degeneration is commonly found, and is due in most instances not to the pericarditis, although generally caused by the same condition as the pericardial inflammation. Death rarely results in pericarditis from this lesion. Independent myocardial degeneration leading to dilatation of the heart, and especially fatty degeneration of the myocardium, is a predisposing or determining factor toward pericarditis. Overaction of the heart may induce pericardial inflammation. Serofibrinous pericarditis is in most instances an evidence of generalized bacteremia. Chronic adhesive pericarditis is a lesion of great frequency, often impossible of diagnosis, and in itself of very little clinical significance or importance. Serious symptoms arise from adhesive pericarditis only when the myocardium itself is seriously diseased, either concomitantly or quite independently. The signs usually cited as characteristic of pericardial synechia develop only when mediastinal inflammation or adhesions of marked degree are present in addition to the pericarditis.

NORMAL AUSCULTATORY DIFFERENCES BETWEEN THE SIDES OF THE CHEST.

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Differences at the Bases Behind. From those who are concerned to make the earliest possible diagnosis of pulmonary tuberculosis we hear more and more loudly each year the command to study with all possible care and attentiveness the auscultatory differences between corresponding portions of the two sides of the chest, as well as between different portions of the same lung. The slightest variation in pitch, quality, intensity, or duration of either inspiration or expiration at any point, as compared with conditions in the corresponding portion of the other lung, is, we are told, a sign to be noted, since it may be one of the factors in a decision affecting the patient's whole future life.

If this is true, and I see no good reason to doubt it, it behooves us to know as much as we can about any differences existing between the auscultatory phenomena in the right lung and those in the left in the healthy chest. It has been clearly recognized, at any rate since Austin Flint's time, and by the great majority of competent practitioners, that the apices of the two lungs differ in a large proportion of healthy persons, the apex of the right being more like that we expect to find in beginning solidification (from any cause) than is the apex of the left. All this is too familiar to need repetition. I wish, however, to call attention to what I believe to be a fairly common difference between the right and the left base posteriorly.

In a series of 250 healthy cases recently examined with reference to this point, I found the difference to which I allude to be present in 67 per cent. In these cases I noticed that the breathing at the left base was notably louder and harsher than that at the right. In the majority of these it had also the quality described by the Germans as "rough." I have been unable to see that age, sex, the time of day, or the amount of food in the stomach had any effect upon this phenomenon; yet I found it sometimes present in the morning and absent in the evening, for reasons unknown to me. It seems to be distinctly less common in persons under twenty years of age.

I have nothing of importance to say as to the cause of this difference. Thinking that it might be due to the amount of gas in the stomach, I inflated a patient's stomach with air and auscultated the backs of the lungs before and after the inflation, but I could not see that any considerable effect was produced by this procedure. One might conjecture that the slightly lower position of the diaphragm upon the right, or the greater impingement of

the heart upon the left than upon the right lung, might have some influence in producing the auscultatory differences to which I have referred, but these are unverifiable conjectures, and seem to me practically valueless. I have not found any differences in percussion or palpation accompanying the phenomena to which I have just referred.

Obviously, the only importance of the sign which I have described is that in case it is confirmed and established by the observations of other physicians it will slightly complicate our process of reasoning in regard to the soundness or unsoundness of the lungs as based upon physical examination. It may be that we shall have to make allowance for the differences to which I have just referred, as we have been in the habit of doing with the differences between the two apices.

Differences in the Lateral Recumbent Position. Almost every good clinician is familiar with the fact that when the patient is lying upon his side the restriction of the motion of this side of the thorax leads to differences in the phenomena obtained by palpation, percussion, and auscultation in the lower lung, as compared with the upper. Hence we are all of us chary of drawing conclusions from what we hear in a patient's chest in the lateral recumbent position. We all prefer to examine the patient, either sitting up or lying on his face, whenever one of these positions can be assumed without serious inconvenience. But I wonder how many of us would be able to state, promptly and correctly, just what are the differences between the upper and the lower sides of the chest of the patient lying upon his side. As a matter of permanent and accurate record, it seems to me worth while to formulate as accurately as we can the knowledge which we all of us now possess in a more or less inchoate form.

With this in view, I recently examined fifty normal chests with the subject in the lateral recumbent position. Briefly stated, the differences are as follows: (*a*) On palpation, increased tactile fremitus on the lower side; (*b*) on percussion, a combination of dulness with a tympanitic quality on the lower side; (*c*) on auscultation, an increase in the intensity of the spoken and of the whispered voice, with a slight prolongation of expiration and a raising of its pitch.

Summarily stated, these signs amount to the indications of a slight degree of condensation of the lung, such as we see in the upper part of a chest when a pleural effusion is present below.

It is perhaps also worth noting that in patients, both of whose lungs contain approximately an equal number of rales when the patient is examined in the sitting position, lateral decubitus increases the number of audible rales on the lower side.

THE PRACTICAL VALUE OF SPINAL PERCUSSION IN DISEASES OF THE MEDIASTINUM.[1]

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Curiously enough, the systematic study of normal vertebral resonance and of its pathological modifications has never come into general vogue as a routine method of physical examination, so that to most clinicians the technique of vertebral percussion, in comparison with the more familiar procedure of what may be termed mural percussion of the thorax, is virtually a closed book. Despite this indifference on the part of medical men in general, several noteworthy contributions have been published in recent years on the subject of spinal percussion and the various tonal changes thereby demonstrable in diseases of the thoracic and abdominal cavities. Twelve years ago, von Korányi[2] drew attention to the fact that in pleural effusions displacement of the posterior mediastinum abolished the normal resonance of the lower spinal vertebræ and produced a triangular patch of dulness extending therefrom to the unaffected side. The more recent investigations of Grocco[3] and his followers along these lines are too well known to call for more than passing comment. Nagel and de la Camp,[4] in a recent series of experimental and clinical studies, have clearly demonstrated the value of percussing the spinous processes as an aid to the diagnosis of enlarged bronchial and mediastinal lymph nodes. Ewart's careful original work[5] on percussion of the vertebræ and its correlation with dorsal percussion also forms a most important contribution, and will be referred to at more length in a subsequent paragraph, as will von Korányi's elaborate studies on the vertebral percussion zones.[6] In passing, it is of interest to recall the fact that almost half a century ago Piorry[7] advocated "pleximetry of the spine," and described in this connection a vertical band of universal vertebral *dulness!* Obviously, this cardinal error in acoustics is sufficient to dismiss this investigator's conclusions from serious consideration.

The subject matter of this communication deals primarily with the technique of spinal percussion and the application of this method of research to the clinical investigation of mediastinal lesions, which in the cases under consideration consist chiefly of enlarged bronchial and mediastinal lymph nodes met with in connection

[1] Read by title at the meeting of the Association of American Physicians, Washington, D. C., May 11 and 12, 1909.
[2] Handbuch d. spec. Path. u. Therap., 1897, iv, 717.
[3] Rivista crit. di clinica med., 1902, iii, 13.
[4] Jahrbuch f. Kinderheilk., 1908, lxviii, 46.
[5] Lancet, 1899, ii, 261.
[6] Ztschr. f. klin. Med., 1906, lx, 295.
[7] Traité de Plessimétrisme, Paris, 1866.

with tuberculosis and other pleuropulmonary infections, and which also include several examples of neoplasms implicating the middle and posterior mediastinal spaces. Lesions such as these, as will be explained presently, affect the percussion sound over the thoracic vertebræ, and with this segment of the spinal column alone is the present paper concerned.

The normal osteal percussion resonance of the spine as a whole, elicited by rather *forcible percussion* of the vertebral spinous processes, is characterized by a sound of well-sustained volume and duration, of relatively high pitch, and of a peculiar osteal quality—the last two criteria being judged by their comparison with the pitch and the quality of healthy pulmonary resonance. Vigorous percussion blows, then, bring out this type of resonance along virtually the entire length of the spinal column, from the nape of the neck to the coccyx, for, when sharply struck, an individual spinous process transmits the impact to neighboring vertebræ, above and below, and the vibrations set up in this manner induce a sonorous tone whose dominant element is resonant. This synchronized and concerted plexímeter action of the vertebræ under the action of a sharp percussion blow provokes a general resonance of the spine, despite the possible existence of certain acoustic factors, which, were they not smothered, so to speak, by the loud osteal tone, might damp less powerful vertebral vibrations, and thus modify the percussion findings along areas of the spine that abut upon both normal and morbid structures of the thoracic and abdominal cavities. To all intents and purposes, the spinal column acts as a long plexímeter, and the vibrations thereupon set up by the percussion impact travel not only up and down the vertebræ and laterally along the ribs, but also to some extent are conducted toward and even into the organs and other structures of the thoracic and abdominal cavities.

In contrast to the foregoing, by *gentle percussion* it is possible to distinguish, in the healthy subject, differences in the percussion sounds over certain areas of the spine, which differences are explained partly by the resiliency of the segment percussed, and partly by its proximity to various pre- and para-vertebral structures. The former condition, that is, the inherent vibratory properties of the spine itself, is an all-important factor in determining the character of the percussion findings over the different spinal segments. The dulness ordinarily afforded by the cervical region is to a large extent attributable to such a cause, for, aside from other physical influences, this part of the spinal column is anatomically unfitted to generate a resonant sound: to the plexímeter finger it presents a short, uneven, concave column of bone, composed of compact vertebræ tipped with short bifid spines, held close and rigid by a tense ligamentous network, and unprovided with articulating ribs for the lateral conduction of vertebral vibrations. On the

other hand, the thoracic zone seems peculiarly well adapted to yield resonance on percussion: to the plexímeter finger it presents a long, even, convex segment of bone, unaffected by disturbances of tension referable to excessive flexion, and made up of large vertebræ having long, stout spines and costal paths for the transmission of vibrations. The influence of contiguous structures in modifying the spinal percussion sound is to be considered in intimate correlation with the anatomical factors just mentioned, and in pathological conditions of the mediastinum such influences must, naturally, dominate. In the cervical region, the tympanitic effect of the prevertebral air passages and gullet is effectually negatived by the structural peculiarities of the spine here existing, so that the sound remains dull so long as the percussion impact is gentle, though a tinge of laryngotracheo-oral tympany is appreciable on forcible percussion while the subject's mouth is open. The thoracic vertebræ, which abut anteriorly and laterally upon the lungs, are consequently clear and resonant on light percussion, and they become increasingly hyperresonant as the force of the blow becomes greater. The fact that this normal resonance of the thoracic spine is definitely modified by certain mediastinal diseases demonstrates the advantage of spinal percussion as a routine clinical procedure. Lesions of this intrathoracic space, it should be remembered, may impinge against the spine so closely as to damp its vibrations most effectually, without affecting in the slightest degree the percussion sound of the thoracic wall proper.

The Normal Spinal Percussion Zone. Percussion of the vertebral spinous processes by the method subsequently to be described delimits a vertical zone overlying the twelve thoracic vertebræ and affording percussion sounds which differ acoustically according to the situation of the vertebra percussed. In health, percussing the successive spines downward from the vertebra prominens, the sound is frankly dull, the tactile resistance exaggerated, and the pitch high over the first three, sometimes four, thoracic spines, below which level the remaining eight or nine thoracic spines afford clear, low-pitched resonance tinged with a distinctive osteal quality. With a hammer and ivory plexímeter one can furthermore distinguish certain circumscribed patches of impaired resonance along this stretch of uniform resonance elicited by ordinary mediate finger percussion. Thus, Ewart[8] lays great stress upon the isolated dulness of the fifth thoracic spine, which modification he considers an invariable normal finding, attributable to "the cessation at the level of the fifth vertebra of the resonant influence of the trachea and to the replacement of this resonant influence by the dulling influences due specially to the infratracheal glands." That dulness in this situation is clearly demonstrable in instances

[8] Loc. cit.

of bronchial glandular tumors is a well-recognized fact, but it is not easy to understand how it is possible for a group of normal-sized bronchial lymph glands thus locally to damp the vertebral vibrations, despite the lost resonance of the windpipe at this level. The authority just quoted also speaks of "postcordial" impaired resonance over the sixth and seventh thoracic spines; of "left auricle" dulness over the eighth and ninth; and of "posthepatic" modified dulness over the tenth and eleventh. While recognizing the academic interest of the foregoing refinements of the percussion sound, their practical service as diagnostic aids must appear questionable.

In an elaborate study of vertebral percussion von Korányi[9] has mapped out the entire spine, from occiput to coccyx, into five separate zones, each of which, he asserts, furnishes distinctive percussion findings. In brief, these zones, with the sounds therein obtainable with identical percussion technique, are as follows:

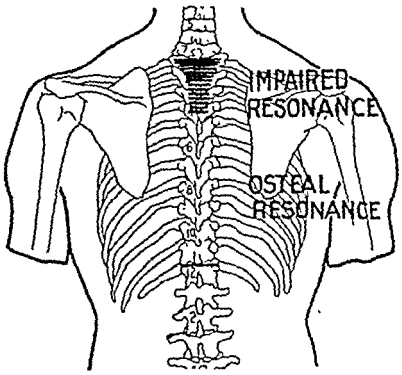

FIG. 1.—Normal percussion zones of the thoracic spine. Impaired resonance and increased tactile resistance from the first to the third or the fourth spinous process; clear osteal resonance from the fourth or the fifth to the twelfth spine. (The figures have been prepared from Keen's Clinical Charts, published by P. Blakiston's Son & Co.).

(1) first to seventh cervical, dulness; (2) first to fourth thoracic, dulness blending below with impaired resonance; (3) fifth to eleventh thoracic, clear resonance; (4) twelfth thoracic to fifth lumbar, dulness progressively diminishing until tympany appears over the last lumbar spine; (5) sacrum and coccyx, tympany. These take into account the whole extent of the spinal column, and constitute a somewhat more formidable and complicated percussion map than is necessary for routine clinical work. Of the five different zones outlined, but two (the cervical and the thoracic) can be depended upon invariably to show definite findings, and of these two the cervical, being so far removed from the predominant influence of the pulmonary structure, can safely be dispensed with, save under

[9] Loc. cit.

most exceptional circumstances. The lumbar and sacral segments, whose physiological tympany is so materially interfered with by the dulling action of solid matter within the bowel, do not enter into the consideration of mediastinal affections, and in the investigation of lesions of the abdominal organs which should impair the tympany of this lower vertebral zone, the percussion findings of this level must needs be interpreted with great caution, for the reason just specified.

The accompanying illustration (Fig. 1) shows the two spinal zones corresponding to the crevical and thoracic vertebræ, to be reckoned with in dealing with the diagnosis of mediastinal disease.

Technique of Spinal Percussion. In order to obtain interpretable data from spinal percussion one must observe consistently certain details of technique relating chiefly to the subject's posture and to the method of eliciting the percussion sounds. As a preface to remarks on this score, it should be stated that it is useless to attempt the accurate percussion of a diseased spinal column, the pleximeter action of which is interfered with by various inflammatory processes and deformities affecting the osseous structure and its vibratory properties. The latter are also altered in thoraces having such an unnatural convexity of the vertebral extremity of the ribs that the spine lies in a deep gutter walled on either side by a high costal ridge. Excessive rigidity and overdevelopment of the musculature (especially of the vertebral, vertebrocostal, and vertebroscapular groups) are two additional bars to successful percussion of the spine, and it is quite obvious that a generous layer of fat upon the back must have a similar effect.

The subject's posture must be such as perfectly to relax both the vertebral muscles and the interspinous ligaments, to insure which the patient should sit upon a cushioned stool (or upon a chair, facing its back), with the trunk inclined forward so that the crossed arms repose lightly upon the knees, and with the head bent forward at an angle of about 50 degrees from the spinal column. In this position mistakes due to undue spinal rigidity from musculoligamentous tension need not be feared. On the contrary, when the subject sits upon a hard stool or stands upright the spinal column must necessarily be subject to the increased tension inseparable from the proper maintenance of the given posture. It is, of course, manifestly impracticable to percuss the spine with the subject lying in lateral decubitus.

Mediate finger percussion, as ordinarily practised, is the most satisfactory method of percussing the spine, the stroke being directed so as to fall upon the dorsum of the pleximeter finger applied individually to the tips of the spinous processes, beginning at the first thoracic vertebra and thence percussing downward toward the hyper-resonance of the lumbar segment. The stroke should be delivered somewhat sharply, but, and this detail is all important,

with moderate force, the exact degree of which can be determined only by personal experience. Too much force may bring out a confusing hyper-resonance which wholly masks the dulness actually existing; too little force may fail to demonstrate anything tangible. Some idea of the proper degree of force to be used can be learned by percussing, with strokes progressively increasing in strength, the spinous processes at the level of the inferior scapular angle, until the spine emits a clear, sonorous, resonant tone and at the same time conveys to the pleximeter finger a sense of moderate resiliency at the moment the percussion impact occurs. With precisely the same technique percussion of the cervical and upper two thoracic spines should, under normal conditions, produce high-pitched dulness with unmistakably increased tactile resistance. In judging the character of the vertebral percussion findings, the resistance appreciated by the pleximeter finger is a far surer guide than the sound produced by the percussion impact, in view of which point it follows that mediate finger percussion is preferable to instrumental percussion with a hammer and ivory or metal plate.

Summary of Case Reports. The twenty mediastinal cases herewith presented to illustrate the utility of spinal percussion were selected from some 300 odd records of examinations made during the last two years in routine hospital practice. For the sake of clearness, the individual percussion findings in these cases have been diagrammed, with a brief *résumé* of the clinical histories. In every instance the diagnosis of the mediastinal affection was verified by an *x*-ray examination, as a supplement to the evidence presented by the patient's symptoms and by the more familiar mural physical signs. The last two sources of information were sufficient to warrant almost certain clinical opinions in eleven of the cases (Cases I, III, VII, VIII, IX, XII, XIII, XIV, XV, XVI, XIX), and in these the vertebral percussion findings were confirmative of the other objective symptoms, save in a single instance (Case XII), in which, despite a pertinent clinical picture and a convincing radiogram, careful percussion of the upper part of the thoracic spine afforded nothing more than a modification of the osteal resonance. This discrepancy, for lack of a more tangible cause, seems best explained by assuming that the subject's concomitant emphysema, which was typically developed, must have exerted a disproportionately hyper-resonating influence on the spinal vibrations. This influence, it may be added, was readily demonstrable by percussing the vertebral column immediately below the strip of interscapular impairment. The predominant effect of emphysematous hyper-resonance upon the spinal percussion sound is clearly shown by the chart of Case IV, which suggests a zone of excessive resonance at the levels of the second and third thoracic spines (Fig. 5). Much the same peculiarity is also shown, though somewhat less conspicuously, by Cases VI, X, and XII,

but only in the last instance did the physical signs spell hypertrophic emphysema (Figs. 7, 11, 13). It is conceivable, however, that the dulling effect of an intrathoracic solid mass may be more or less

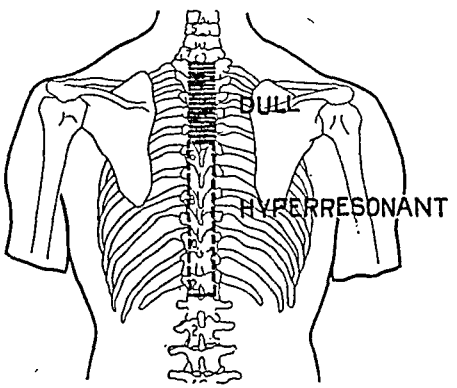

FIG. 2

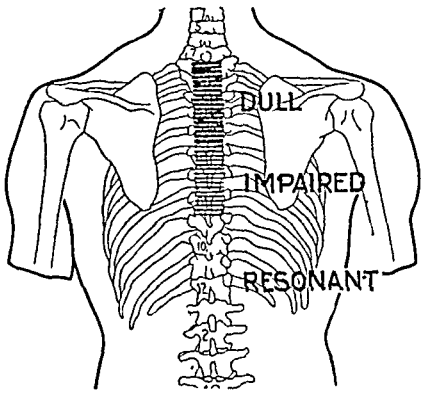

FIG. 3

FIG. 2.—Case I. Mary S., tuberculous lymphadenitis of the cervical and tracheobronchial glands; persistent venous congestion of the upper thorax and neck; cough, dyspnea, and symptoms referable to pressure upon the trachea, primary bronchi, superior vena cava, and pneumogastric nerves. *Absolute dulness and greatly increased plexímeter resistance from the first to the fifth thoracic spine, below which percussion yields loud sonorous hyperresonance.*

FIG. 3.—Case II. James S., tracheobronchial glandular enlargement; tuberculous infiltration (disseminated) of both lungs; small serofibrinous effusion in the left pleural cavity. *Frank dulness from the first to the fifth thoracic spine, with impaired osteal resonance over the vertebral column from the sixth to the eighth thoracic vertebra. Grocco's paravertebral dull triangle not demonstrable.*

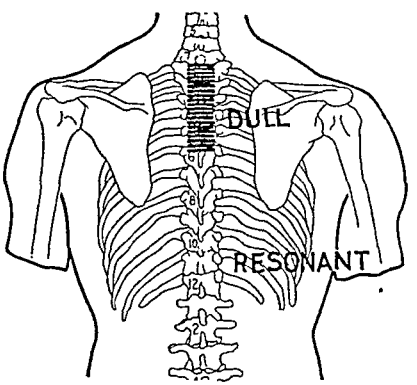

FIG. 4

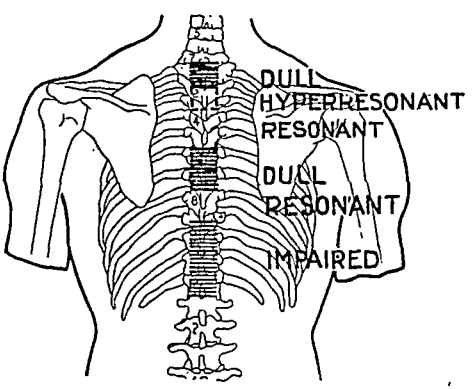

FIG. 5

FIG. 4.—Case III. Rebecca S., disseminated lymphadenitis of the cervical, supraclavicular, and mediastinal glands; postural congestion and tumefaction of the head, neck, and arms; dilatation of the superficial veins of the upper thorax, neck, and face; pressure symptoms relating to the trachea, bronchi, superior vena cava, and inferior laryngeal nerve. *Uniform osteal dulness from the first to the fifth thoracic spine, with clear resonance over the remaining vertebræ of the interscapular zone.*

FIG. 5.—Case IV. Elizabeth S., mediastinal and cervical adenitis; chronic ulcerative phthisis; bilateral pulmonary emphysema. *Striking dulness over the first thoracic spine; hyperresonance over the second and third spines; dulness over the sixth and seventh spines; impaired resonance from the ninth to the twelfth spinous process of the thoracic zone.*

negatived by the interposition of areas of compensatory vesicular dilatation affecting the inner and posterior pulmonary borders—which assumption, if true, must largely invalidate the real usefulness of spinal percussion in emphysematous subjects.

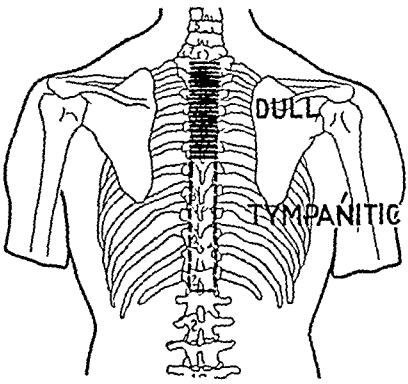

FIG. 6

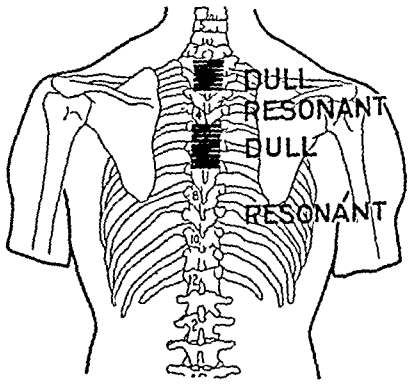

FIG. 7

FIG. 6.—Case V. Annie K., enlarged bronchial glands; chronic ulcerative phthisis; chronic adhesive pleurisy of the left side. *Uniform dulness from the first to the fifth thoracic spine, with clear, low-pitched tympanitic resonance of osteal quality from the sixth to the twelfth process.*

FIG. 7.—Case VI. John D., enlarged bronchial glands; chronic fibroid phthisis. *Dulness over the first thoracic spine; resonance over the second and third spines; dulness from the fourth to the sixth spine; then downward to the twelfth, normal osteal resonance.*

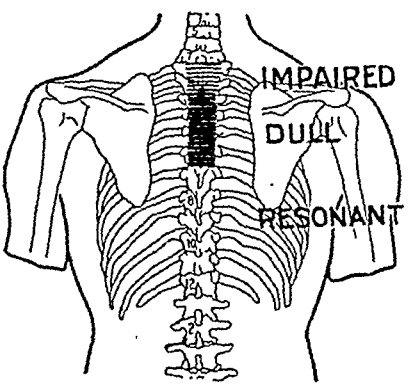

FIG. 8

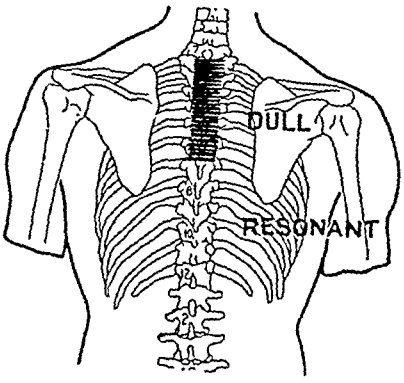

FIG. 9

FIG. 8.—Case VII. William R., mediastinal adenitis; tumefaction of the face and neck; substernal and interscapular pain; dyspnœa; cough. *Impaired resonance over the first and second thoracic spines; striking dulness and increased plexímeter resistance over the third, fourth, and fifth spines; normal osteal resonance over the remainder of the thoracic zone.*

FIG. 9.—Case VIII. Ella E., tuberculous adenitis of the cervical, axillary, and tracheobronchial glands; bilateral dry pleurisy; permanent jugular engorgement; postural congestion of the face and neck; dyspnœa, cough, and mucopurulent sputum; constant pleural pain. *Dulness from the first to the sixth thoracic spine, with normal osteal resonance thence downward to the base of the thoracic zone.*

To sum up the advantages of spinal percussion in the study of obscure mediastinal lesions, it may be concluded that the method is distinctly useful, particularly in that class of cases presenting

indefinite pressure symptoms and mural signs, and that it also has a certain corroborative value in the face of a more tangible clinical

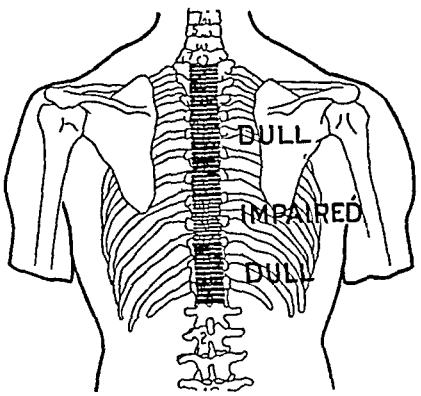

FIG. 10

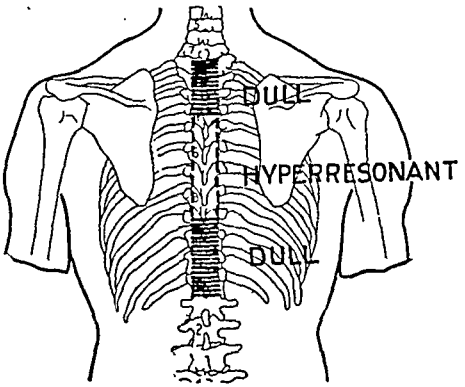

FIG. 11

FIG. 10.—Case IX. Margaret T., enlarged bronchial glands; small effusion in the left pleural cavity; paroxysmal attacks of dyspnœa, dry cough, vertigo, and syncope; substernal and epigastric pain; bulging of the left parasternal region; enlargement of the superficial veins of the upper thorax. *Frank dulness and greatly increased plexímeter resistance from the first to the sixth thoracic spine; impaired resonance over the seventh and eighth; dulness (Grocco's) from the ninth to the twelfth spines.*

FIG. 11.—Case X. John K., enlarged bronchial glands; cardiac hypertrophy; right lateral cardiac displacement; small serofibrinous effusion in the left pleural cavity; constant thoracic pain, suppressed dry cough, dyspnœa, and shallow respiration. *Uniform dulness from the first to the third thoracic spines; vertical (spinal) limb of Grocco's triangle extends from the ninth to the twelfth spines; intermediate spinous processes (fourth to eighth) afford loud hyper-resonance.*

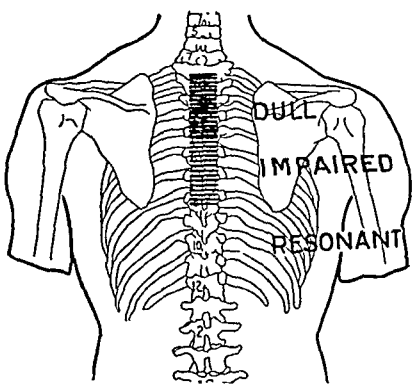

FIG. 12

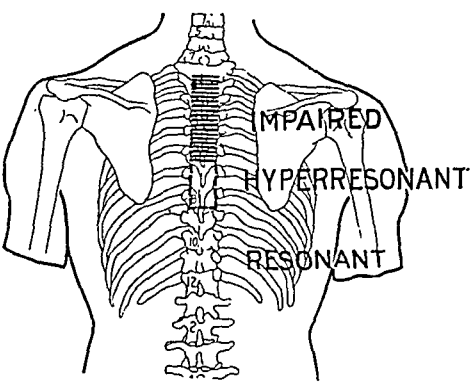

FIG. 13

FIG. 12.—Case XI. James H., enlarged bronchial glands; chronic bilateral adhesive pleurisy. *Uniform dulness from the first to the fourth thoracic spine, with decidedly impaired resonance over the lower four spinous processes of the interscapular region.*

FIG. 13.—Case XII. George R., enlarged bronchial glands; bilateral disseminated pulmonary fibrosis and hypertrophic emphysema; cardiac dilatation; habitual cyanosis; paroxysms of precordial pain, orthopnœa, deep cyanosis, and vertigo. *Impaired resonance (but not absolute dulness) from the first to the sixth thoracic spine, with hyperresonance over the next two processes of this zone.*

picture. In the former category belong those very frequent examples of enlarged bronchial and mediastinal glands, so fre-

quently consecutive to tuberculosis and to other specific infections, in which vague and puzzling pressure symptoms and physical signs are the rule. In such instances the discovery of a dull vertebral

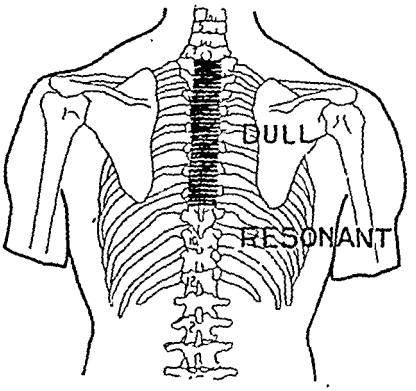

FIG. 14

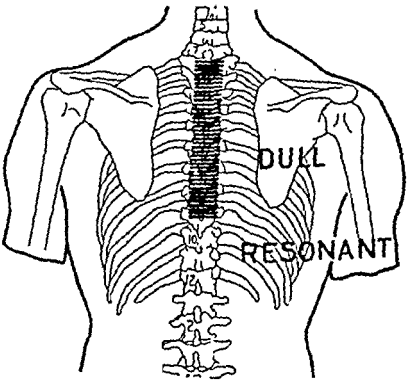

FIG. 15

FIG. 14.—Case XIII. William C., mediastinal neoplasm; bronchial compression; recurrent laryngeal nerve irritation; dyspnœa, tracheal tugging, laryngeal voice, substernal pain, "brassy" cough, acute paroxysms of cyanosis, orthopnœa, and cough excited by physical exertion and by forward inclination of the trunk. *Uniform dulness from the first to the eighth thoracic spine, the greatest degree of impairment being over the upper six vertebral processes.*

FIG. 15.—Case XIV. Robert B., mediastinal gumma; postural congestion and tumefaction of the face, neck, and arms; dilatation and unnatural tortuosity of the superficial veins of the head, trunk, and arms; pressure symptoms referable to the œsophagus, trachea, bronchi, superior vena cava, and vagus; bulging of the sternum and of the left parasternal region; downward and left lateral cardiac displacement. *Uniform absolute dulness from the first to the eighth thoracic spine, with conspicuous exaggeration of pleximeter resistance of the entire thoracic zone.*

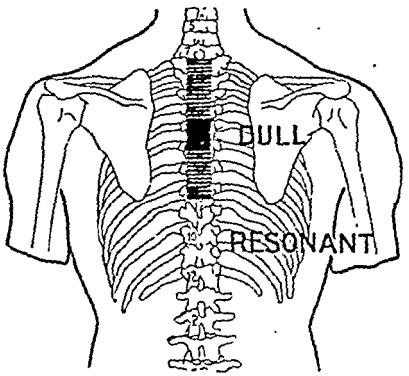

FIG. 16

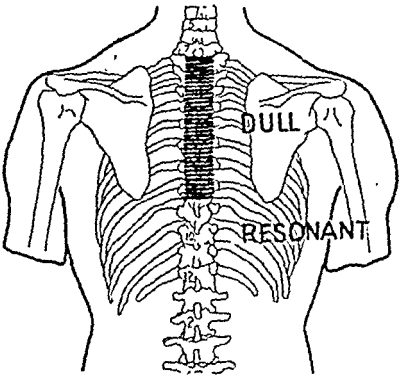

FIG. 17

FIG. 16.—Case XV. John B., fibroid (?) tumor of the middle and posterior mediastinal spaces; downward and left lateral cardiac displacement; compensatory dilatation of the left upper pulmonary lobe; paroxysmal cough, dyspnœa, and shallow respiration; habitual laryngeal voice; constant substernal pain. *Spinal dulness extends uniformly from the first to the eighth thoracic spine, the impaired resonance and increased resistance being especially conspicuous over the fifth and sixth spines.*

FIG. 17.—Case XVI. Edward K., Hodgkin's disease; enlarged mediastinal glands; progressive enlargement of the cervical, axillary, and inguinal glands; symptoms of bronchial and venous compression. *Dulness and greatly exaggerated pleximeter resistance from the first to the seventh thoracic spine.*

strip below the fourth thoracic spine gives a most significant clue, and one ordinarily corroborated by a subsequent *x*-ray examination. Mediastinal neoplasm, aneurysm of the aortic arch, tumor of the

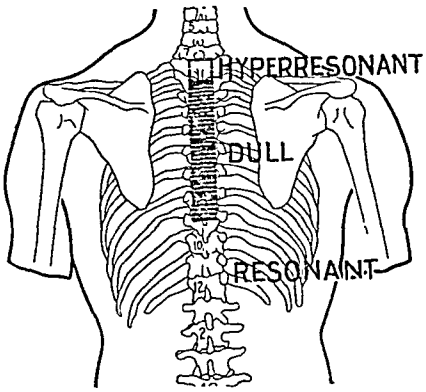

FIG. 18

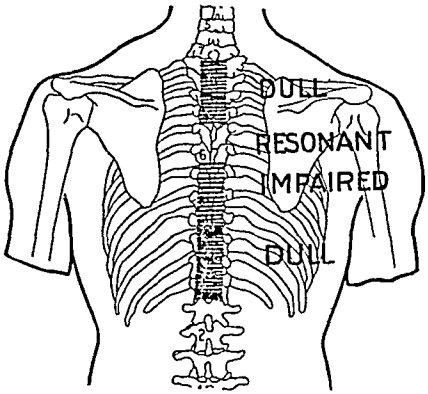

FIG. 19

FIG. 18.—Case XVII. John S., myelogenous leukemia; enlarged mediastinal glands; splenic tumor 51 x 36 cm.; upward displacement of the heart and of the left phrenic dome. *Impaired resonance from the second to the eighth thoracic spine, with an unnaturally high-pitched and clear percussion sound over the first thoracic spine.*

FIG. 19.—Case XVIII. Henrietta S., myelogenous leukemia; enlarged mediastinal glands; splenic tumor 42.5 x 26 cm.; retrodisplacement of heart. *Dulness over the first, second, and third thoracic spines; clear resonance over the fourth and fifth; impaired resonance and increased plexímeter resistance over the sixth, seventh, and eighth; frank dulness from the ninth to the twelfth thoracic spines.*

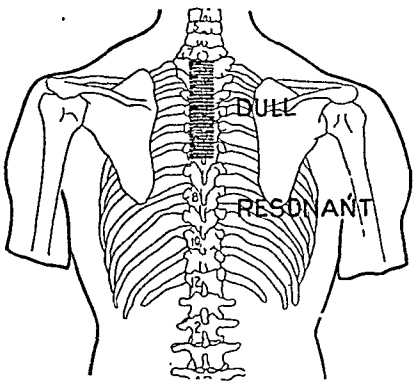

FIG. 20

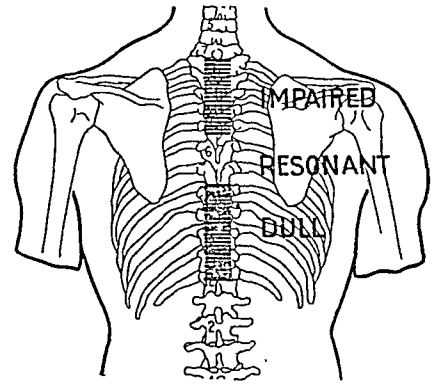

FIG. 21

FIG. 20.—Case XIX. Laura F., myelogenous leukemia; enlarged mediastinal glands; splenic tumor 30 x 22.5 cm.; cough, dyspnœa, and other signs of tracheobronchial pressure. *Impaired resonance and increased plexímeter resistance from the first to the fifth thoracic spine, with clear percussion sound below the latter level.*

FIG. 21.—Case XX. Henry S., lymphatic leukemia; enlarged mediastinal glands; splenic tumor 38 x 21 cm.; enlarged cervical, axillary, and inguinal glands. *Impaired resonance from the first to the fourth thoracic spine; resonance over the fifth and sixth; dulness from the seventh to the twelfth spine.*

œsophagus, atelectasis, consolidation of the lung, and pleural effusion also may encroach backward so as to damp the spinal sonorousness; these lesions should always be taken into account as potential factors of unnatural dulness over the thoracic vertebræ.

RESEMBLANCES BETWEEN THE CLINICAL EFFECTS OF PNEUMOCOCCIC AND MENINGOCOCCIC INFECTIONS.

By Robert B. Preble, M.D.,

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The systematic employment of blood culture methods in all cases of obscure infections has shown that the pneumococcus is frequently the cause of clinical pictures which differ widely from one another and from the commonest effect of this organism, croupous pneumonia. However, careful review of a y large number of these cases will show that there are certain features which recur with great constancy, no matter how much the cases may differ from one another. These phenomena I have come to regard as the specific effects of the pneumococcus, and from them I have been able repeatedly to predict the results of the blood culture. These effects are four in number, and while no one of them is peculiar to the pneumococcic infection, the combination of three of them, much less all four of them, is seen in the course of no other infection except one.

The first of these is the acuity of the onset of the symptoms of infections. Pneumonia is conspicuously a disease of sudden onset, the patient in most instances being able to fix not only the day, but in many instances the hour, and even the minute, when the symptoms began. This is equally true of other forms of pneumococcic infections. The sudden onset of symptoms is, of course, not peculiar to this infection, for there are numerous other organisms which initiate symptoms equally suddenly.

The second effect is the polymorphonuclear leukocytosis, and it, too, is common to many other infectious processes, some of which are also characterized by sudden onset, although there are several of the very acute infectious processes unaccompanied by leukocytosis.

The third effect of the pneumococcus, an herpetic eruption, is much more highly specific, being present in a large, though not constant percentage of the cases, and being common to but two other infections, namely, malaria, a process due to an organism of so widely differing nature that no consideration of it is here necessary, and secondly, meningococcic infection, in which, like the pneumococcic infections, herpes is very common. Moreover, the herpetic eruption occurs usually over the same nerves, one or the other of the lower branches of the fifth cranial nerves, although in either it may occur over any other nerves. The herpetic eruption in the cases of meningitis is not to be referred to the meningitis, for in no other form of inflammation of this membrane

is herpes common, except in those cases which are due to the pneumococcus.

When we consider these three effects, not singly, but in a group, one finds that while there are many infections causing a combination of the first and second and one infection causing the combination of the first and third, there is no other infectious disease in which all three of them occur, except in cases of the so-called epidemic meningitis.

Now, if to these three one other is added, the resemblance between the two processes is not increased by any mere arithmetical, but by a considerable geometrical, progression.

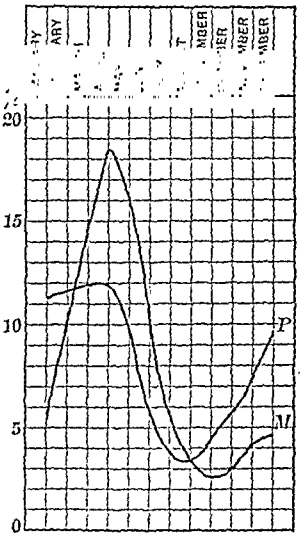
For years the extremely low chloride excretion in the urine in cases of pneumonia has been appreciated and has been utilized both as a means of diagnosis and prognosis, but it is not yet as generally known as it should be that in other forms of pneumococcic infections, such as tonsillitis, endocarditis, meningitis, sepsis without localization, the same extremely low chloride excretion is found. This I regard as a fourth specific effect of pneumococcic infection.

Naturally this has suggested a study of the urine of cases of meningitis as to the chloride excretion. No light upon the subject is thrown by a study of the literature, for as far as I have been able to find no mention is made of it, but in all the cases of epidemic meningitis which I have seen of late not a large number, it is true, have shown the same low chloride excretion which is shown by cases of pneumonia. We find, therefore, that while there are several infectious processes which show combinations of two of the specific effects of pneumococcic infections, there is no other except the meningococcic infection which shows a combination of three, much less one of all four of these effects.

These, however, do not exhaust the similarities between the two infections. There are certain complications which occur with greater or less frequency with pneumococcic infections, such as endocarditis, pericarditis, arthritis, and otitis media, and it is interesting to note that these same complications occur also with the meningococcic infections. Not much stress can be laid upon this, for these same things occur in many other infections. There is, however, one point upon which I should like to lay some stress, namely, that of the endocarditis. It has long been my opinion, based purely upon clinical observation, that endocarditis of pneumococcic origin occurs when the cocci are of low virulence, and this opinion has recently been experimentally confirmed by Rosenau. If, then, the suspicion is correct that the meningococcus is a pneumococcus of lowered virulence, then one would expect to find endocarditis more common as a complication of the epidemic meningitis than it is in pneumonia in the young. Pneumococcic endocarditis as a complication of pneumonia of the old or of those having an antecedent valvular lesion must be excluded from con-

sideration. One must also make allowance for the fact that certain of the cases of meningitis run so acute a course that there is not time enough to recognize clinically a developing endocarditis. Nothing very definite can be said upon endocarditis as a complication of meningitis, because of the lack of data in literature; even in the reports upon autopsies made but few details are stated. I feel sure that the lessened mortality from this disease, such as we seem warranted in expecting from the antimeningococcic serum, will be accompanied by numerous reports of subsequent endocarditis.

The frequency with which meningitis is accompanied by pneumonia may have some bearing upon the question of the relationship of the two organisms, but here again we lack data. Practically all of the early literature upon the question is valueless, and no doubt many of the instances in which the two processes were associated are due to the pneumococcus, which admittedly may cause either or both.



Another point of resemblance between the two diseases is that of their seasonal distribution. The accompanying curves (see chart) show the percentage distribution according to months. They are rather grossly alike, but would probably more closely resemble each other if they were upon anything like a similar number of cases. The pneumonia curve by its smoothness suggests that it is based upon a large series of cases, and this is true, for it is made up from tables including over 600,000 cases, while the meningitis curve is based on but 6000 bacterially confirmed cases.

It was formerly stated that the epidemic meningitis did not end by crisis, one of the most striking peculiarities of the pneumococcic infections, and this was true, but since the introduction of the

Flexner-Jobling serum, this is no longer so, for all who have observed the effects of this serum in many cases report endings as critical as any seen in pneumonia.

Another interesting thing about the epidemic meningitis is that cases occur sporadically separated from all known sources of infection and this, too, though the meningococcus is an organism known to have but little resistance to outside influences. This fact is difficult to explain if one admits that the meningococcus is an independent organism, but not so difficult if one assumes that it is merely a modified pneumococcus, an organism which is practically ubiquitous.

It must be admitted that many, possibly the great majority of the cases of sporadic epidemic meningitis are not truly instances of this disease, but even so, there seems nowhere any disposition to question the occurrence of sporadic cases.

In contrast with these clinical resemblances one finds certain others of clinical differences which are significant, namely, the striking difference in the age distribution of the two diseases, one being conspicuously a disease of childhood, while the other occurs most often in adult life. The second point is the frequency with which the meningitis is associated with a hemorrhagic diathesis, as shown by petechial eruptions. Such a diathesis is not unknown in pneumonia, but it is extremely rare. I can recall but two instances of it, if one excludes the not infrequent cases of early hemoptysis in pneumonia.

It appears to me that the points of resemblance between the two infections are sufficiently striking to warrant serious consideration by the bacteriologists who, without exception, I believe, regard the two causal organisms as absolutely distinct, although admitting that they have many points in common, so many, indeed, that their differentiation is so difficult that much of the early work upon the bacteriology of meningitis and notably that of the nose and pharynx in this disease must be disregarded.

THE DIAGNOSIS AND SURGICAL TREATMENT OF ACUTE PANCREATITIS.

By John B. Deaver, M.D., LL.D.,
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The diseases of the pancreas are, at the present time, being subjected to an ever-increasing amount of clinical observation and to experimental research of the most varied forms. Especially is this true of acute pancreatitis, a disease which in its several varieties

touches upon some of the most important problems with which the abdominal surgeon and the physiologist of today must deal. Acute pancreatitis is a rare disease, although close study reveals the fact that many cases must in former times have been overlooked. It presents difficulties in diagnosis and treatment, and it is but rarely that either the surgeon or the medical man has the opportunity of seeing a large series of cases. I consider myself fortunate in having had the opportunity of observing closely several, and of operating on six cases. These are as follows:

CASE I.—A white male, aged forty-four years, in the early part of 1901 suffered an attack of epigastric pain, lasting three hours and followed by diarrhœa. Pain recurred about every six months, was cramp-like in character, and relieved usually by mustard plaster. From 1901 to 1902 he felt very well, but afterward had pronounced symptoms of indigestion, loss of appetite, vomiting, belching of gas, and paroxysmal pain in the region of the stomach. During the week previous to the last attack the abdomen was distended, there was constipation, and some yellowness of the sclerotics. The diagnosis by his attending physician was intestinal obstruction. Operation showed acute inflammation of the pancreas, which I believe supervened upon a chronic pancreatitis. The gall-bladder was drained, but no stones were found. The patient recovered and is perfectly well at the present time, having had no further trouble referable to the abdomen.

CASE II.—White, male, aged forty-two years, admitted to the German Hospital November 11, 1902.

The patient's past medical history is negative, except for occasional attacks of indigestion. His general health is good, but for the last twenty-five years he has had attacks of pain in the epigastrium lasting three to four hours, and accompanied by jaundice. No history of constipation was noted. Three days before admission he was seized with abdominal cramps, and the next day had epigastric pain and vomiting associated with much belching. Bowels did not move. On admission the patient was suffering with violent paroxysms of epigastric pain, was belching frequently, and vomiting. The vomitus was a clear, green, bile-stained fluid. The patient's bowel movements while in the hospital were clay-colored.

Physical examination showed the presence of slight icterus. The abdomen was markedly distended and tympanitic, the distention being most marked in the epigastric region. The *x*-ray examination showed the excursions of the diaphragm to be somewhat limited, and revealed a shadow in the region of the pancreas. The temperature on admission was 102.8° F., the pulse 104, and the respirations 30. The leukocyte count, November 14, was 8200, and on November 21, 1902, 13,850, with 81.3 per cent. of polymorphonuclears. The stools showed free fat. There was no glycosuria at any time.

The patient appeared to improve somewhat. The pulse dropped

to between 86 and 96 to the minute; the respirations to 24, and the temperature to between 99.6° and 101° F. before the operation.

On November 22, 1902, the patient was operated upon, under ether narcosis. The incision was made through the upper portion of the right rectus. Some fat necrosis of the gastrocolic omentum was found. The gastrocolic omentum was opened, exposing the pancreas, which was found to be diseased, with the head of the gland thickened. Some hemorrhage was present. The pancreas was drained at the site of the hemorrhage with a rubber tube, around which was packed sterile gauze. Death occurred soon after the operation.

Autopsy. The preperitoneal fat and all the fat in the abdomen, omentum, and mesentery was studded with little areas of fat necrosis. The pancreas was much diseased. The tail was gangrenous and surrounded by a reddish, purulent fluid. The head and body of the organ had remained relatively normal. The lesser peritoneal cavity contained much purulent, reddish-yellow fluid, containing broken-down pancreas. The gall-bladder was full of calculi, but it was impossible thoroughly to examine the biliary ducts owing to adhesions and necrosis. About the sloughing gangrenous pancreas there were adhesions; but there was no general peritonitis.

The salient features of this case, to which I shall refer more in detail in the general discussion, are: (1) General abdominal distention, most marked in the epigastrium; (2) the presence of biliary vomiting and frequent belching; (3) the drop in pulse rate, respiration, and temperature, with improvement in the patient's general condition, after the severe initial symptoms; (4) the presence of fat necrosis; (5) the presence of biliary calculi; (6) the involvement of the tail of the pancreas almost exclusively; (7) the presence of free fat in the feces and the absence of glycosuria; (8) the absence of free fluid in the abdomen; and (9) the mistake of not opening the lesser peritoneal cavity through the left loin posteriorly, instead of transperitoneally.

CASE III.—C. K., white, female, aged sixty-one years. Admitted to the German Hospital January 20, 1905.

The patient was admitted with the diagnosis of acute intestinal obstruction, and was operated upon immediately. No history was obtainable. On admission the pulse was 76 to the minute and the temperature was 99° F.

Under ether narcosis, an incision was made in the left inguinal region, in the belief that the sigmoid was at fault. The descending colon and sigmoid were examined and found normal, and the original wound was closed. The abdomen then was opened in the median line, the incision extending from the ensiform to the pubis. The entire intestine was searched for an obstruction, but none was found. At the greater curvature of the stomach near the pylorus a dense mass of adhesions was found, with hemorrhage beneath. The adhesions

were separated and the pancreas was found hard, nodular, enlarged, and the seat of a large hemorrhage. Four pieces of gauze were carried to the head of the pancreas and a glass tube was placed in the pelvis. An antiseptic dressing was applied. The patient died fifteen hours after the operation.

Postmortem. The pancreas was 24 x 8 x 4 cm. in size and weighed 290 grams. It was dark red in patches, and densely adherent to the stomach and the transverse colon. Areas of fat necrosis were found in the surrounding tissues. The gall-bladder contained bile and about fifty stones; the ducts were patulous.

This was evidently one of those extremely acute cases of pancreatic hemorrhage, that is, hemorrhagic pancreatitis, referred to by some authors as fulminant. The operation took place within a day of the onset of the illness, even before fat necrosis had an opportunity to occur, yet the very severity of the disease rendered the prognosis hopeless. Especially to be noted is the diagnosis of acute intestinal obstruction. As will be seen on reading case reports of this disease, this diagnosis is very commonly made—far oftener than the true one. We find in this case also: (1) Fat necrosis; (2) the presence of calculi; and (3) a very slow pulse rate—76—with a temperature of 99° F.

CASE IV.—White, female, aged forty-one years. Admitted to the German Hospital October 17, 1905.

No history of tuberculosis or malignancy was obtained. The patient had always had general good health. Nine weeks before admission she became gradually ill, complaining of pain in the right side of the abdomen referred to the back, epigastrium, and right iliac fossa. Three weeks ago she had severe abdominal pain in the right side of the abdomen, but most severe in the upper portion. The pain was followed by vomiting and was almost constant. Some soreness in the upper abdomen was also noted. The nausea and vomiting had been more or less continued. On admission the patient's abdomen was distended, especially above the umbilicus. Rigidity of both recti muscles was noted, but most marked above the umbilicus on the right side. There was an indefinite mass in the epigastrium, which was moderately tender. On admission the temperature was 100° F., pulse, 76. While the patient was in the hospital the leukocytes varied from 8600 to 32,900, and the hemoglobin from 45 to 68 per cent. Free fat and occult blood were found in the stools.

Operation, October 18, 1905. Under ether narcosis, an incision was made in the median line above the umbilicus; the stomach was found distended and pus escaping through the foramen of Winslow. The wound was closed and a second incision made along the right costal margin, exposing the lower margin of the liver adherent to the stomach. The adhesions between the stomach and the liver were separated, and the gastrohepatic omentum was found to be thickened and œdematous. An aspirating needle was passed through the thick-

ened gastrohepatic omentum in the direction of the lesser peritoneal cavity, and blood and pus were obtained. A free incision was then made, allowing the escape of considerable pus. A rubber tube, surrounded with gauze, was then placed in the lesser peritoneal cavity. The gall-bladder was searched for but not found. After the operation the patient developed a septic temperature. The leukocytosis continued, and she left the hospital in poor condition, with a discharging sinus. She died after some weeks of continued sepsis.

In this case we may note particularly: (1) Gradual onset of the illness, indicative of a subacute suppurative process rather than an acute necrotic one; (2) continued vomiting; (3) low pulse rate—76—before operation; and (4) an indefinite mass in the epigastrium.

CASE V.—White, male, aged twenty-seven years. Admitted to the German Hospital March 18, 1908.

One year before admission the patient had four or five attacks of abdominal pain, accompanied by jaundice. Two and one-half weeks before admission he had a severe attack of epigastric pain, accompanied by nausea and vomiting. The pain continued up to the date of admission, with frequent exacerbations. It started in the epigastrium, and was referred all over the abdomen, and to the back and shoulders. The patient has been jaundiced more or less ever since the onset of this attack.

On admission, the patient was jaundiced, the respiratory excursions were limited, and the respirations short. The liver extended from the sixth interspace to two fingers' breadths below the costal margin in the mamillary line. There was slight epigastric fulness and spasticity of both recti muscles in this region. Some tenderness was noted over the entire epigastrium, and it was quite marked over Mayo Robson's point. The pain continued without relief up to the time of operation. The patient's temperature on admission was 98.4° F., and remained febrile during the entire course of the illness, but only for about three days after operation, with a maximum of 100.4° F. The pulse on admission was 88.

The operation was performed on March 21, 1908, under ether narcosis. An incision was made through the right rectus. The gall-bladder was found adherent to the colon and omentum, and contained calculi. Posterior to the stomach there was a soft fluctuating mass about the size of two fists, pushing the stomach forward. A finger placed in the foramen of Winslow found this to be in the position of the pancreas. The gall-bladder was walled off with gauze pads and aspirated; 40 c.c. of mucopurulent fluid was removed. This was sterile, as shown subsequently by culture. The gall-bladder was opened and four large and twenty-four small stones were removed from it and the dilated cystic duct. A tube drain was introduced into the gall-bladder, and it was sewn to the peritoneum. The choledochus was found to be patulous. The wound was closed and the patient placed

on his right side and an incision made in the left loin, extending down 7 cm. from the costal margin, and just external to the outer border of the erector spinæ. Within the fatty capsule of the kidney there was much fat necrosis. An abscess was evacuated in the location of the pancreas, and about half a liter of bloody purulent fluid escaped. The cavity was drained with a large rubber tube and two pieces of gauze.

The patient made an uneventful and practically afebrile recovery. The drain was left in the gall-bladder eleven days, and that in the posterior incision for several weeks, although the drainage gauze in this incision was removed in six days. The discharge from this wound was found to be very irritating to the skin.

The points of interest here are: (1) The slow pulse and afebrile course after operation; (2) the presence of biliary calculi; (3) the presence of fat necrosis around the abscess cavity; and (4) the irritating character of the pancreatic discharge.

CASE VI.—H. M. N., a white male, aged thirty-nine years. Admitted to the German Hospital December 28, 1907.

The case, which has been reported by Dr. Jurist,[1] was one of gangrenous pancreatitis, in which following a posterior incision purulent and necrotic material was removed from the region of the pancreas. Ten days after the operation. and again a week later, large pieces of necrotic and gangrenous pancreas were discharged through the wound. Thereupon the patient's condition improved markedly, and eventually he made a perfect recovery. He now enjoys excellent health, his metabolism apparently is undisturbed, and he has no glycosuria.

Of special importance in this case are: (1) The primary shock with a slow pulse; (2) extreme prostration thereafter, with signs of profound toxemia; (3) the operation in the second stage; and (4) the use of the posterior incision alone.

In discussing this series of cases, it will be admitted that in each case there was an acute pancreatitis. It is true that they are of various types. Thus, in Case II we have an example of a slowly progressing type, of the hemorrhagic variety. This may perhaps be considered one of those cases in which the inflammation preceded the hemorrhage by some time, as the latter does not furnish the changes most marked pathologically, nor is the course that of an acute hemorrhagic case. Case IV represents the ultra-acute hemorrhagic pancreatitis of Mayo Robson's classification, in which the hemorrhage is the primary factor. The rapid course and extreme disorganization of the gland itself make this apparent. This represents that class of cases which is least promising for any form of surgical intervention—indeed, when the pancreatic disintegration has

[1] AMER. JOUR. MED. SCI., 1909, cxxxviii, 180.

reached the stage which it had in this instance, it is almost hopeless to expect a recovery. Cases IV and V represent the acute suppurative variety of pancreatitis: not a localized abscess in the pancreas, but a more or less diffuse suppuration with involvement of the omental bursa. Case VI is of the gangrenous type. Doubtless this commenced as an acute necrosis also, probably hemorrhagic in type, which involved nearly all of the gland, as is shown by the extent of the gangrene. The subsequent well being and perfect assimilation of the patient would lead us to suppose that a portion of the head of the pancreas was entirely unaffected by the disease.

ETIOLOGY. As to the etiology of acute pancreatitis much has been written, largely from the standpoint of experiments. Clinically several factors have been mentioned. Many authors have called attention to the fact that people in whom acute pancreatitis is found are, as a rule, of middle age and inclined to stoutness. As regards the age, this would hold true in my cases, with the exception of Case V. In Cases II, V, and VI the patients were, however, very well developed, and not given to excess of adipose tissue. In Case VI, there was the history of a diabetic parent, perhaps only a coincidence, but nevertheless rather striking.

Opie[2] has called attention to the association of various forms of pancreatitis with gallstones, especially those situated in the choledochus. In Cases II, III, and V, I found gallstones, but in no case obstructing the choledochus. In Case IV the gall-bladder was not found; it was so bound down by adhesions that the search for it in the presence of so much free pus, would have been dangerous. In Case VI the bile ducts naturally could not be inspected; but it seems reasonable to assume that stones were present, in view of the many attacks of pain resembling biliary colic.

Thus, in six cases of acute pancreatitis, four (67 per cent.) had gallstones. In two additional cases personally communicated to me, which have recently occurred in this city, gallstones were not present. It is noteworthy that in the three cases in which gallstones were actually found there was no stone in the choledochus. I have a number of times found chronic pancreatitis associated with a stone in the common duct.

My experience, as regards biliary calculi, coincides with that of most authors. The investigations of Opie have already been mentioned. The only investigator of prominence who disagrees with these conclusions is Truhart,[3] who, in the analysis of a large number of cases, found that a smaller percentage of patients with acute pancreatitis had gallstones than those who died from other causes.

Fitz, Opie, and most American and British authors have inclined to the belief that the causative factor in acute pancreatitis is most often some form of infection, while Chiari and his school incline to

[2] Diseases of the Pancreas, 1905.

[3] Pankreas Pathologie, Wiesbaden, 1902.

the theory of a chemical autodigestive process. The experimental results of various investigators have differed greatly on this subject. While Opie, Fitz, and others, as mentioned, give great prominence to the factor of infection, Truhart, in an analysis of 74 cases of acute pancreatitis, found that in only 14 could microörganisms be demonstrated. Naturally this refers to the pancreatitis itself, for when pus has once formed and an abscess is present, this is likely to become secondarily infected. Then again, the fact that microorganisms cannot be shown by culture does not eliminate them as causative factors. In one case (V) Bacillus coli as demonstrated in the pus.

It seems reasonable to assume, however, when gallstones are present and not in the choledochus, that whatever causative action they may have had has been exerted by the concomitant infection. A curious feature in this connection is that in the case mentioned the apparently purulent fluid contained in the gall-bladder, showed no bacteria, while the colon bacillus was found in the pancreatic abscess.

Case III is an example of those in which commonly no bacteria are found, and seems to be a case of primary hemorrhage into the pancreas.

PATHOLOGY. The pathology of acute pancreatitis has been the subject of much discussion, and bears an intimate relationship to the question of etiology. So far as my records go, there was nothing to throw any light on the presence of an autodigestive processes, which so many German authors, notably Doberauer[4] and Guleke[5] have brought into the foreground as the initial process in acute pancreatitis. On the whole, I incline to the theory of infection rather than that of primary chemical change in the pancreas. So many substances act as stimuli to the disintegration of the pancreas by so-called autodigestion that it seems quite likely that bacteria can also do this. And, indeed, Hekma[6] furnished experimental proof of this fact.

Sailer and Speese[7] found that a toxic substance was present in the blood of dogs suffering from acute pancreatitis experimentally produced by the injection of sterile oil into the pancreatic duct followed by ligation of the duct. Williams and Busch[8] found "anatomical and experimental evidence that made it seem probable that some cases of acute pancreatitis may be caused by regurgitation of duodenal contents into the diverticulum of Vater, the way having been opened by the passage of gallstones."

DIAGNOSIS. The diagnosis of acute pancreatitis is the most important question that confronts us. Even a cursory examination of

[4] Verhand. Deut. Ges. f. Chir., 1906, xxxv, 280.
[5] Arb. aus d. chir. Klin. d. K. Univ., Berlin, 1906.
[6] Quoted by Doberauer, Arch. f. Phys., 1904, p. 334.
[7] Trans. Assoc. Amer. Phys., 1908, xxiii, 540.
[8] Jour. Med. Research, 1907.

recorded cases will show that in 90 per cent. the correct diagnosis has not been made, except at operation or autopsy. Indeed, even at operation the condition has been overlooked in not a few instances (as in a case lately reported as one of a series by Noetzel[9]).

The symptomatology, therefore, must first be considered. Fitz[10] gives us the best concise statement of the classical symptoms: "Acute pancreatitis is to be suspected when a previously healthy person, or sufferer from occasional attacks of indigestion, is suddenly seized with violent pain in the epigastrium, followed by vomiting and collapse, and in the course of twenty-four hours, by a circumscribed epigastric swelling, tympanitic or resistant, with slight rise of temperature."

The pain, as a rule, is severe, and generally referred to the epigastric region. It was so in all of my cases (II, III, V, VI) in which this is recorded. It has been asserted by some that it is more often to the left of the median line, and while this may be a point of some value when it is present, the absence of localization of the pain to the left side should not influence us in the least in estimating the importance of this symptom. The existence of severe epigastric pain was noted in several cases reported by Mayo Robson, in the two reported by Bornhaupt,[11] in a case reported to me in a personal communication by Dr. Chase, of Philadelphia, and in several of those reported by Doberauer and Noetzel, and those of Murray[12] and Bell.[13] On the other hand, in several of the cases reported by Doberauer, the pain was referred to in a more general way as being abdominal. Indeed, in one case operated by Noetzell, the patient spoke of the pain being in the lower abdomen to such an extent that the operator made his first incision for a supposed pelvic condition.

There is never any doubt concerning the severity of the pain wherever located. It practically always calls for the guarded use of opiates, although these are often without avail. This was true in Case VI, of my series in which the pain was agonizing, and but little affected by several hypodermic injections of morphine. Associated with the pain, but a far less constant symptom, is epigastric tenderness. In many cases it is but slightly marked, although careful examination will usually elicit it. It becomes more marked if the case progresses to suppuration, especially with the formation of a mass, which is practically always tender. In the same way epigastric muscular spasticity may occur as the result of the local condition.

Soon after the onset of the pain there is vomiting—a practically constant symptom. The vomiting, in some instances, lasts but a few hours or perhaps a day—as in Case V; and in others it becomes almost uncontrollable and progressively worse. This is a diagnostic

[9] Beitr. z. klin. Chir., 1908, lxxv, 735.
[10] Quoted by Robson and Cammidge, Diseases of Pancreas, 1908.
[11] Deut. med. Woch., 1908, xxxiv, 1306.
[12] Trans. Amer. Surg. Assoc., 1902, xx, 219.
[13] Ibid., 1904, xxii, 103.

sign of but little value, as it is common to most acute intra-abdominal conditions. Attention has also been called to the fact that it is, as a rule, biliary in character. Indeed, vomiting, especially when persistent, is one of the most misleading symptoms of the disease under consideration, as it often serves to make a diagnosis of intestinal obstruction seem the correct one. I have, however, never noticed fecal vomiting, which is always present in the late stages of intestinal obstruction.

Persistent and uncontrollable belching and hiccoughing are marked symptoms of acute pancreatitis. They are often sufficiently severe to draw the physician's attention to the possibility of some trouble near the diaphragm, and to help to a correct diagnosis. They were especially noted in Case VI of my series, and were far more troublesome than the vomiting itself.

Collapse in the acute onset of pancreatitis is often very marked. In some of the cases, operated and unoperated upon, it may pass very quickly on to a fatal termination. In others, the patient recovers from it sufficiently to last for a few days, in very rapid cases, or even recovers therefrom entirely when a localized pancreatic suppuration supervenes. Fitz and others have called attention to the marked cyanosis which occasionally accompanies this collapse, and it has been referred to as a diagnostic feature. Its cause is still unexplained. It was not noted in any of my cases, but was a feature in the case personally reported to me by Dr. Chase.

A possibly important symptom in the onset of acute pancreatitis is the frequent presence of a slow pulse even during the collapse, and until a septic condition supervenes. Indeed, it may even persist under these circumstances. I have had several instances of this fact sufficient to convince me of its *occasional* significance. Thus, in Case II, the pulse was never very rapid and dropped even to below 100 when the other symptoms abated. In Case III, an ultra-acute one, the pulse on admission was 76. References to the suppurative and less acute cases, Nos. IV and V, show a comparatively low pulse rate. In Case VI, during the primary agonizing pain and collapse the pulse was about 80 to 90 per minute, even when the temperature dropped below 96° F. In this instance, however, the pulse became very rapid and feeble as soon as the marked pancreatic toxemia had a chance to manifest itself.

So far I have been discussing practically only the symptoms immediately associated with the very onset of the disease. If the patient survives the initial shock or hemorrhages, as the case may be, certain other conditions make themselves manifest. There are evidences of intestinal paresis. Besides the vomiting and belching already spoken of, there is frequently in the first two or three days, interference with the passage of gas and feces. After several days repeated enemas will usually give quite copious and bulky stools. Associated with these conditions there is intestinal and abdominal

distention, with corresponding distress. Very often this seems to be largely colonic, affecting principally the transverse and descending colon, the small intestine being involved to a lesser extent.

Slight jaundice, as noted in Case VI for forty-eight hours, and in Case II and Case V (with cholelithiasis) is of frequent occurrence in pancreatitis. As a diagnostic sign it would serve only the purpose of calling our attention to some lesion in the upper abdomen.

The formation of a more or less well-defined epigastric mass, apparently deep, usually somewhat tender, and often to the left of the median line, is very suggestive of pancreatitis, when found in conjunction with some of the other significant symptoms. Formerly, when cases were kept under observation longer, because of failure to diagnosticate them and reluctance to operate, except in the face of very definite indications, this was more often seen than it is now, when the tendency is to operate somewhat earlier. Case VI showed this feature most markedly, that is, the presence of a deep indefinite resistant area in the left epigastrium. Some suggestion of it was noted in Case IV. In the case reported to me by Dr. Chase this feature was most marked.

Naturally the presence of a tumor, such as this, can hardly be expected in those instances of acute pancreatitis which quickly terminate fatally. The swollen, engorged, or hemorrhagic pancreas itself is hardly ever palpable. The hemorrhage in and about the pancreas itself scarcely ever gives us such a considerable mass that palpation is possible, yet even this must be considered within the range of possibility.

Finally, there is to be mentioned that occasional occurrence of dulness or of impaired resonance and breath sounds over the lower lobe of either lung, usually the left. This occurred in Case VI of my series, and led to a diagnosis of pleurisy as a complication. The condition was present at the time of operation, but then was not interfered with. I believe it was due to infection through the diaphragm.

While the diagnostic points in acute pancreatitis are not uniform, there are enough, in at least a certain percentage of cases, to enable us to diagnosticate the condition.

Differential Diagnosis. The differential diagnosis is a matter of some difficulty in most cases. In the majority of instances in which an incorrect diagnosis has been made, it has been that of acute intestinal obstruction. It is not difficult to see why this is so. In both conditions we have the sudden onset, the violent, cramp-like pain, a slow pulse in the beginning, some distention which gradually increases, and vomiting following the pain, with stoppage of gas and feces. The similarity is heightened by the fact that in pancreatitis the distention is largely colonic, and this often leads to a suspicion of an obstruction low down. At the onset of the disease it may be impossible to differentiate the conditions, yet the uncertainty should not

last very long. In the first place, it is rare to find shock, with occasional slow pulse and cyanosis in intestinal obstruction. The pain rather, where extremely severe, causes the patient to look blanched and exhausted. Moreover, it is, as a rule, localized to some point in the lower abdomen, rather than in the epigastrium, as in pancreatitis. Yet, as I have mentioned in discussing the symptom of pain, atypical locations thereof in pancreatitis are not entirely unknown. As the case progresses for some hours the distinction becomes slightly plainer. The vomiting in obstruction is one of the cardinal symptoms. It is persistent, severe, and becomes progressively worse. The vomitus becomes foul in a short time. In pancreatitis the vomiting is bilious in practically every instance; after a short time it becomes less frequent. Far more distressing is the hiccoughing and belching associated with pain. These seem to be much dwelt upon in the case histories of pancreatitis, and were illustrated to a marked degree in Case VI of my series. They are not so prominent or striking in cases of obstruction. In pancreatitis the distention is generally colonic and not of high grade. In obstruction it may or may not be localized to the colon, is more marked and more quickly progressive. In pancreatitis the ileus may be absolute at first, yet often high enemas result in passage of gas and true bowel movements. In acute obstruction we can, by the same means, obtain only emptying of the lower part of the larger bowel, and the passage of flatus, if it occurs at all, is very limited.

In pancreatitis the development of epigastric pain occurs frequently and often early, while in obstruction we have practically never a localization of these signs to the epigastrium. Slight jaundice often develops in pancreatitis; in obstruction it is absent. The rapid development of free fluid in the abdominal cavity would point to pancreatitis. If the case should progress for some days, we have in pancreatitis the gradual appearance of signs of omental bursitis, an upper abdominal lesion, associated with a severe toxemia, but with subsidence of the signs of ileus, vomiting, etc. In obstruction there is also a toxemia from the absorption of stagnated bowel contents, and the signs of intestinal stasis plainly manifest themselves if continued; finally there are fecal vomiting, increasing signs of peritonism, marked distention, and tympany.

The mistake of diagnosticating a case of acute pancreatitis as obstruction was made in Case III. With the increased understanding of the subject which we now have, and perhaps a slightly more extended study of the case (which was operated upon at once on admission), the diagnosis, I believe, could perhaps have been properly made. It is a most noticeable feature of all the more extended series of cases of pancreatitis reported, that in the earlier ones the diagnosis was but rarely made, while in the later ones seen by any one man it was practically always strongly suspected, if not definitely decided upon.

Acute gastroduodenitis has been mentioned as possibly to be con-

fused with pancreatitis. It must, indeed, be a severe type of this illness which would simulate the graver lesion. Indeed, it seems hardly more than necessary to consider the symptoms of each to arrive at a diagnosis, should this be the differential question.

Biliary colic, with or without slight jaundice, would give us many points of similarity to the initial pain of pancreatitis. At the time of the onset of the pain a distinction might be impossible. Gall-stone colic is far more common than acute pancreatic lesions are, and they may be associated, as in Case V, so that the physician would think of that condition first. As a rule, the location of the pain to the right of the median line, its radiation, and the failure of other pancreatic symptoms to supervene should guide us correctly. Yet it is to be remembered that in pancreatitis the location of the pain is variable, and radiation to the back and shoulders has been mentioned as at times occurring.

The diagnosis would be more difficult when we have a perforative peritonitis as the result of some lesion in the biliary passages. This condition would more closely resemble other varieties of perforative peritonitis of the upper abdomen, and will be grouped with these in the discussion thereof.

Other conditions referred to as possibly simulating acute pancreatitis have been poisoning, impacted ureteral calculus, etc. In these the course of events, the other and distinctive signs, and in the former the history, should soon serve to put us on the right track

Thrombosis of the mesenteric vessels, practically never diagnosticated except at operation, may give us symptoms of ileus and peritonism, very likely to simulate acute pancreatitis.

Acute appendicitis has been the diagnosis of several cases of pancreatitis. It does not seem to me that these conditions are sufficiently similar to cause the mistake to be made often. Appendicitis is distinctly an inflammatory condition of the lower abdomen; its symptomatology is well marked and typical in most cases, and its signs with few exceptions are not those of an upper abdominal lesion.

Next to a diagnosis of acute obstruction in those cases of pancreatitis not recognized, there is that of an acute perforative peritonitis of the upper abdomen, be it of stomach, duodenum, or bile ducts. In the first two conditions we can generally get a history which would be something of a guide to us. When the biliary system is the seat of perforation, the history may be misleading rather than a guide to us. In all these conditions the sudden agonizing pain in the epigastrium would at once point to a serious lesion of the upper abdomen.

TREATMENT. The treatment of acute pancreatitis is one of the most important subjects which confronts the surgeon. The comparative rarity of the condition, the different phases it may assume, and the varying degrees of its severity, make the treatment of first importance.

But a few years ago pancreatitis, except when progressing to

suppuration, or the formation of a palpable mass, was considered to belong to the field of medical treatment alone. Surgical intervention was considered hazardous and almost useless. Several cases have been reported as recovering without operation, in which there were apparently the symptoms of an acute pancreatic lesion. I have seen this occur in acute pancreatitis and in acute exacerbation of chronic pancreatitis. Needless to say, recovery under these circumstances, is rare; indeed, it may truthfully be stated that the chance of such a non-operative recovery is comparatively slight.

The medical treatment of acute pancreatitis has been aptly compared to a similar procedure in acute appendicitis. The latter affection was beyond the range of surgery many years after its pathology was well known. It may be admitted then that the treatment of an acute pancreatitis is by surgery only. This being so, there are three important features to be considered as regards operation:

1. At what stage of the illness shall we operate?
2. What method of approach to the seat of disease shall be adopted?
3. The technique after reaching the pancreas or the pancreatic abscess.

1. *The stage at which operation should be undertaken* in acute pancreatitis varies with the variety of the disease. In a fulminating ultra-acute case, the rapid progress of the patient from bad to worse may make early operation necessary. Operation should not be undertaken in the state of primary shock, following the onset of the distinctive symptoms of pancreatitis. Cases in which this has been done have almost invariably come to a fatal termination. The tendency among surgeons who have investigated the question, and have had the opportunity of seeing a number of cases, seems to be to operate comparatively early.

Nevertheless, I am convinced that this rule does not always hold good. Thus, in Case VI, which terminated as a gangrenous pancreatitis, with sloughing of almost the whole organ, it is almost certain that surgical intervention in the first few days would have ended in the patient's death. Our delay, with stimulation, allowed us to operate upon a patient in somewhat improved condition, and to make use of localizing signs to approach the pancreas by a much more favorable route than it would have been possible to adopt had immediate operation been undertaken. It must be understood, then, that while in cases becoming rapidly and progressively worse operation may be imperative, it is generally of advantage to give the organism an opportunity to prepare itself, so to speak, for a still greater tax upon it. Moreover, when we delay until an abscess extending into the loin has formed, we are able to approach it through the loin. Had operation been deferred in Case III, the result could not have been more unfortunate. The formation of a mass in the upper abdomen, or the occurrence of dulness and tenderness toward the

flank, would not only make our diagnosis more certain, but give us a safer route by which to approach the diseased area than through the peritoneum.

2. *The Method of Approach to the Seat of Disease.* The routes to the pancreas may be grouped under two main heads: (1) Those which involve the traversing of the general peritoneal cavity, and (2) the extraperitoneal route by the loin incision.

1. When there is a beginning acute pancreatitis, when the localizing symptoms are all epigastric, when there is a tumor palpable anteriorly, giving a tympanitic note on percussion and a sense of resiliency on palpation, and when there is doubt concerning the diagnosis, we are compelled to make our incision upon the anterior aspect of the abdomen, and to traverse the general peritoneal cavity in order to finally reach the pancreas. Once in the general peritoneal cavity, we may approach the pancreas itself either (*a*) through the lesser omentum, (*b*) through the gastrocolic omentum, or (*c*) through the transverse mesocolon. As a general rule, the anterior or intraperitoneal incision, if I may so call it, has been adopted by almost all operators. It has one serious disadvantage, that is, the risk of infecting the general peritoneal cavity. Its advantages are the free exposure of the operative field, opportunity for radical surgery, and the opportunities which it offers for the establishment of adequate drainage. The choice of a path above or below the stomach must depend upon circumstances. When we wish to secure an extra path for drainage, tampons and drains must be carried both above and below the stomach.

2. The extraperitoneal route, practically, as for exposure of the kidney, allows us to approach the pancreas, and especially the tail thereof, without entering the general peritoneal cavity. The chief advantages of the extraperitoneal route when feasible cannot be questioned, except possibly by a few enthusiasts who, by preference, open appendiceal abscesses and remove ureteral stones through the peritoneal cavity. Naturally it is only applicable in those instances in which we have a distinct indication for it, that is, the appearance of symptoms pointing to the localization of inflammatory exudate or pus in the loin. By it we get good drainage for the pancreas and the omental bursa. Its disadvantages are, however, many. We get no free exposure of the parts and radical surgery is impossible. We can drain, that is all. Gallstones or concomitant gastric lesions, etc., are entirely inaccessible by this method. Cases III and IV of my series were operated upon by the anterior incision, and correctly so, as in each case the localizing symptoms demanded the opening of the greater peritoneal cavity. In Case V, after the operation for gallstones had been carried out, the postpancreatic collection was evacuated by the loin. Thus, there was avoided the danger of peritonitis, and at the same time, the underlying biliary condition was adequately dealt with.

In this connection I may mention the fact that in some of the

reported instances of acute gangrenous or suppurative pancreatitis, drainage has been instituted by both the anterior and posterior routes. In Case VI we had an omental bursitis with distinct localizing signs in the left loin. Here it was deemed better to undertake the unmistakably less serious extraperitoneal operation, rather than to ignore the symptom which clearly indicated our correct avenue of approach to the seat of the lesion. I do not believe that we should have achieved an equally favorable outcome by any other operative procedure. The wound drained freely, the pancreas, as it sloughed, was discharged without difficulty, and our only unsatisfactory feature was the impossibility of obtaining a full exposure of the wound when dressing it.

3. *The Technique after Reaching the Pancreas.* Whether we have to deal with a pancreatitis still in the presuppurative or hemorrhagic stage, or whether pus has already formed, we have but one cardinal indication for treatment—the establishment of adequate drainage.

Should we find an acute hemorrhagic pancreatitis, or an acute gangrenous pancreatitis, having approached the organ by the intraperitoneal route, our only possible procedure locally is to apply tampons and drains freely to the organ itself, going either above or below the stomach. I believe that tube drainage is always essential. Gauze alone will do more to prevent than to establish drainage.

In a previous article I have stated it as my belief that, when we find an engorged and inflamed pancreas, it is expedient to incise the capsule and to apply our drains to this exposed surface. I have since then had no further experience with a case requiring this treatment. Certain recent investigators, however, have come to the conclusion that this incision into the organ itself is unnecessary. Noetzel, especially, who has written the most recent resume on the subject takes this stand. He brings strong support for his argument from his own experience and that of others. In the present state of our knowledge it must be admitted that the question is still open.

It is practically never possible satisfactorily to resect the pancreas for inflammatory lesions, and this is, indeed, unnecessary. Drainage gives free exit to the debris.

Should there be any free fluid in the peritoneal cavity it will be necessary to see to its removal. It should not be forgotten that in this pancreatic exudate we have toxic material often sufficient to cause death. In this connection it is interesting to note that in one of the first cases of this kind reported, that of Halsted, the evacuation of the free fluid in the peritoneal cavity was the only thing attempted. The patient recovered, and while it may be true, as Körte has remarked concerning this case, that the patient recovered in spite of and not because of the operation, yet it is doubtful if recovery would have taken place with any added toxins for him to overcome.

The peritoneal fluid may gravitate toward the pelvis. In several cases in which this was marked, tube drainage of the pelvic cavity,

with the semi-erect position of the patient, has been successfully adopted.

It is almost unnecessary to mention the fact that gauze drainage in all these cases under discussion must be supplemented by the use of tube drains, preferably rubber ones of large caliber. Drainage by gauze tampons alone often defeats its own object, the saturated gauze acting only as a plug and not as an outlet.

When there is a pancreatic abscess, or an omental bursitis and peripancreatitis, free drainage either by the anterior or the posterior route is the prime essential. When pus is present it is of still more importance than under any other circumstances to use free drainage by tubes, and not to rely on tampons alone.

Some question has arisen as to the treatment of an accompanying cholelithiasis found at the time of an operation for acute pancreatitis. I believe that when the patient's condition warrants it, we should correct also the underlying and really primary biliary condition, and so avoid any probability of again having to perform a serious laparotomy. Often, as Noetzel has pointed out, the gravity of the pancreatic condition and the weakness of the patient will prevent us from thus completing our operation. And in several reported cases, such as that of Stieda, the leaving of biliary calculi has apparently been no hindrance to the complete recovery of the patient from the pancreatic condition. If there is any doubt in the operator's mind as to the advisability of dealing with the accompanying cholelithiasis, he had better defer. Two operations and recovery are preferable to one operative procedure with a fatality.

The preparatory treatment for operation must evidently be very brief in most cases of acute pancreatitis. It does not offer us great possibilities. We can do little to help the patient to stand the shock of surgical intervention beyond the use of the ordinary stimulants, etc. Speed and the avoidance of shock and chilling on the table play a far more important role in helping the patient to ultimate recovery.

Postoperative treatment is the same as in all other serious laparotomy cases, with but few added features. Drainage should be left in until it has very evidently fulfilled its functions—until there is nothing more to drain.

Recently, Wohlgemuth[14] has, by experiment and also clinically, shown that by placing patients who have been operated upon for pancreatitis upon a typical antidiabetic diet we can so alter the pancreatic secretion that it becomes less active and irritating. This fact seems to be abundantly confirmed by other observers. Not only this, but by the same use of diet, the healing of both recent and old pancreatic fistulæ is accelerated. The results in this direction have been truly remarkable. This is perhaps the only postoperative treatment that differs in any way from that of the other suppurating intra-abdominal lesions and abdominal sinuses.

[14] Berl. klin. Woch., 1907 and 1908.

THE PATHOLOGICAL RELATIONSHIPS OF GASTRIC ULCER AND GASTRIC CARCINOMA.

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The following report is based on the study of specimens from gastric and duodenal resections and excisions for ulcer and carcinoma, by Drs. W. J. and C. H. Mayo, from January 1, 1905, to April 1, 1909. In five of the cases the material was obtained at autopsy from patients on whom gastro-enterostomies had been done for ulcer or carcinoma, and from whom no material had been removed at the operation. These autopsies, however, were all made within one hour after the death of the patient; consequently all the material was quite fresh when placed in fixatives. The routine examination consisted of the study of fresh material, sectioned and stained by the method of one of the writers[1]. Blocks of tissue were then fixed in Zenker's fluid and Flemming's chromo-osmo-acetic fluid and in 10 per cent. formaldehyde. In some instances additional blocks were fixed also in absolute alcohol. The gross specimen was then prepared by Melnikow's modification of Kaiserling's method. The gross specimens were photographed by the method of Wilson and Andrews,[2] either fresh or after fixation. The photomicrographs of sections herewith shown were made from hematoxylin-stained specimens. In these no attempt has been made to show fine detail, which has been sacrificed to a study of the distribution of the cells.

The total amount of material studied comprised specimens from 218 cases. Eight of these were from the duodenum, and were all simple ulcers. The remaining 210 were from the stomach. Of these, 47 were ulcers without suspicion of carcinoma; 2 were sarcomas, 2 adenomas, and 1 a diverticulum. Of the remaining 158 cases from the stomach, 5 were ulcers with enough microscopic appearance of aberrant epithelial proliferation to place them in the doubtful class as possible transition cases. Of the remaining 153 cases, which were undoubted carcinoma, 109 (71 per cent.) presented sufficient gross and microscopic evidence of previous ulcer to warrant placing them in a group labelled "carcinoma developing on preceding ulcer." Eleven other cases (7 per cent.) showed considerable evi-

[1] Wilson, L. B., A Method for the Rapid Preparation of Fresh Tissues for the Microscope, Jour. Amer. Med. Assoc., 1905, xlv, 1737.

[2] Stereophotography of Pathological Specimens: Some Improvements in Technique and New Apparatus, Jour. Med. Research, 1908, xvii, 487 to 494.

dence of precedent ulcer, but not sufficient to warrant placing them in the previous group. In 33 cases (22 per cent.) there was relatively small or no pathological evidence of precedent ulcer.

It is unnecessary to review the enormous literature of this much discussed subject. For years the pendulum of opinion swung back and forth, and it has been only within the last decade that sufficient material from early cases has been collected to give a clear understanding of the facts. The reports on specimens removed at operation during that period have practically settled the question as to the very frequent occurrence of gastric carcinoma on the site of previous ulcer. Our excuse for offering these cases at present is to place them on record as one more bit of evidence to clear up a misconception which has done much harm in the past, and which still exists, as is shown by the attitude of the author of the most exhaustive recent work on cancer.[3]

PROTOCOLS. Case No. 22,826 (Fig. 1). This specimen is from a woman, aged twenty-six years, who for nine years had had some stomach distress, some gas, occasional vomiting, and eructations. For the last eight weeks she had had some loss of strength, had lost in weight (20 pounds), and had vomited occasionally. Stomach analysis showed a total acidity of 65, free hydrochloric acid 50, lactic acid absent, and blood absent. Operation revealed a chronic ulcer of the lesser curvature. Fig. 2 shows the scar tissue and the eroding base of the ulcer within which are no epithelial inclusions. The character of the lesion deep down below the overhanging border, just where the mucosa comes in contact with the basement membrane, is shown in Fig. 3. Here are numerous groups of epithelial cells cut off by the products of inflammation.

Case No. 22,020. The specimen in this case is through the pylorus of a man, aged fifty-one years, who for three years had had some stomach distress with gas, vomiting, etc. For three months he had had some loss of strength and loss of weight (15 pounds), with severe pain. The stomach analysis showed a total acidity of 56, free hydrocloric acid 30, lactic acid absent, blood absent. This case much resembled the preceding one. Fig. 5 shows the eroding mucosa with swollen epithelial cells in the overhanging border of the ulcer. Fig. 6 from near the base of the mucosa shows several small groups of epithelial cells which are segregated from the rest of the mucosa, as in Case 1.

These two cases show how in chronic gastric ulcers in which no carcinoma is demonstrable there already exist isolated areas of epithelium which is under conditions favorable to its aberrant proliferation.

Case No. 18,088 (Fig. 7). This specimen is from the pyloric half of the stomach from a man, aged forty-five years, who had only

[3] Williams' Natural History of Cancer, Wm. Wood & Co., 1908, pp. 279 to 280.

mild symptoms of so-called dyspepsia until nine months ago, when he began to have marked gastric distress, vomiting blood, gas, eructations, loss of appetite, loss of strength, and loss in weight (60 pounds). The stomach analysis showed a total acidity of 50, free hydrochloric acid 12, lactic acid absent, blood present. Operation showed multiple ulcers of the lesser curvature. Three of these were carcinomatous. Fig. 8 is a section from the overhanging border of the ulcer showing the least amount of carcinoma. Fig. 9 is a section from deeper down in the tissues showing the isolated groups of epithelial cells proliferating and infiltrating.

Case No. 18,867 (Fig. 10). This specimen is from a woman, aged sixty years, who for twenty-five years had had more or less severe stomach symptoms, distress, vomiting, gas, etc. For the last six months she had had considerable loss of strength, loss of weight, and severe persistent pain. Analysis of the stomach contents showed a total acidity of 60, free hydrocloric acid 45, lactic acid absent, blood absent. Operation revealed a carcinoma on an ulcer of the lesser curvature. Fig. 11 is from the ulcerating portion of the stomach lesion. Fig. 12 is of a section showing the proliferation of the epithelium without infiltration. Fig. 13 is from a section showing the true carcinomatous character of the lesion.

Case No. 16,525 (Fig. 14). This specimen is from a male, aged forty-six years, who for seven years had had considerable stomach distress with vomiting and eructations of gas. For the last seven months he had had loss of strength and appetite and had lost 45 pounds in weight, being now quite emaciated. The stomach analysis showed a total acidity of 42, with a free hydrocloric acid content of 37, lactic acid present, and blood present. On operation there was found a carcinoma on the border of an ulcer covering a greater portion of the lesser curvature. Fig. 15 is of a section from the ulcerating border showing cross sections of distended glands with round cells between. Fig. 16 shows the bases of glands clipped off by scar tissue. Fig. 17 shows active proliferation in segregated epithelium. Though strongly suggestive of carcinoma, one would hesitate to diagnosticate this section, since the field is obscured by the round cell infiltration. Fig. 18, however, shows typical scirrhous cancer, that is, the inflammation has here subsided and the fibrous tissue has increased around the islands of proliferating epithelium. These four sections are all from the border of the ulcer, but in successive microscopic steps away from its centre.

Case No. 15,681 (Fig. 19). This specimen is from a man, aged thirty years, who has suffered from gastric distress, nausea, vomiting, and gaseous eructations for five years. During the last seven months he had had marked loss of strength, with a loss of thirty pounds in weight. Stomach analysis showed a total acidity of 62, free hydrochloric acid 32, lactic acid absent, and blood present. Operation revealed a carcinoma on an extensive ulcer of the lesser curvature,

FIG. 1

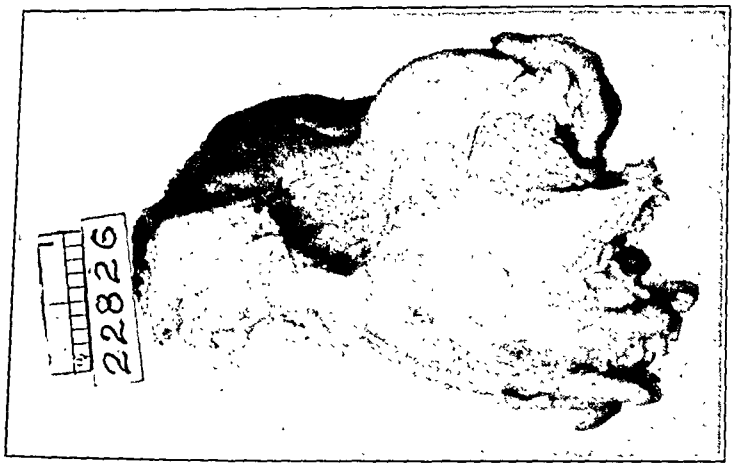

FIG. 2

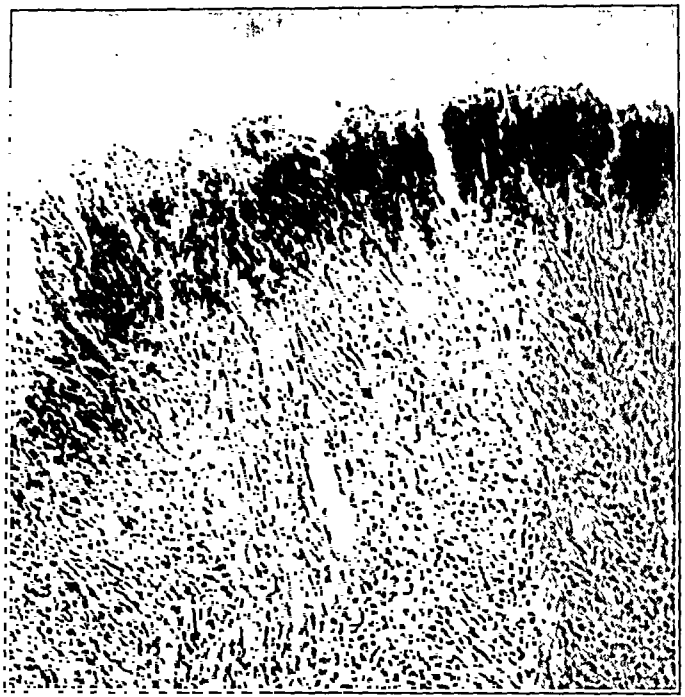

FIG. 1 (Case No. 22,826).—*Stereogram of a chronic ulcer of the lesser curvature of the stomach. Note the overhanging mucosa, the perforation of the muscularis, and the large mass of adhesions.

FIG. 2 (Case No. 22,826).—Photomicrograph of the base of the ulcer. (× 100)

* These pictures were originally shown as stereograms but are here reproduced as single pictures only.

FIG. 3

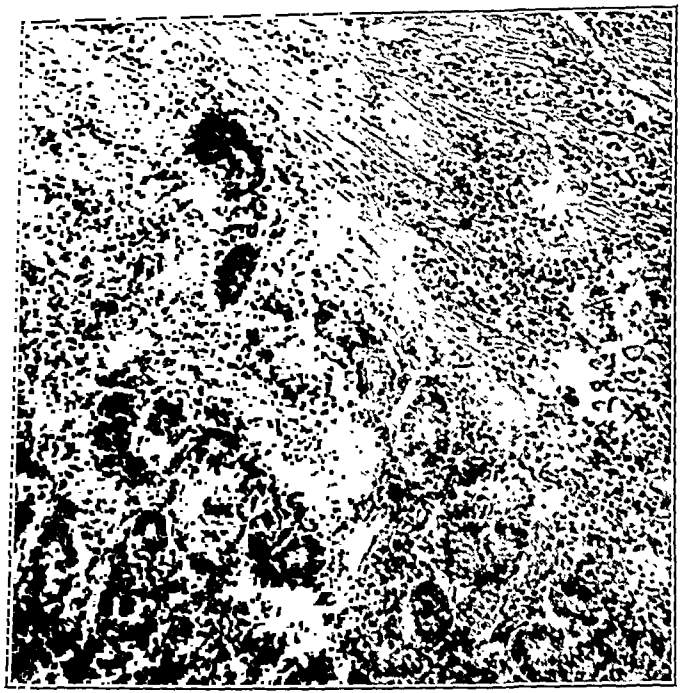

FIG. 4

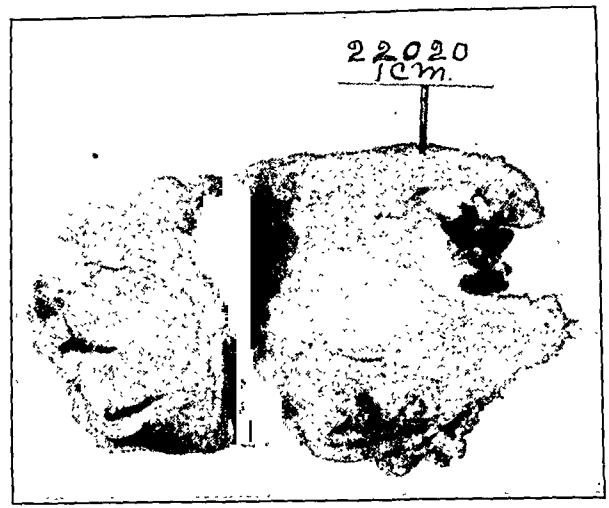

FIG. 3 (Case No. 22,826).—Photomicrograph showing groups of normal epithelium (bases of glands) cut off by products of inflammation deep under the overhanging border of the ulcer. (× 100)

FIG. 4 (Case No. 22,020).—Stereogram of a section through the pylorus, showing the eroded mucosa of a chronic ulcer.

Fig. 7

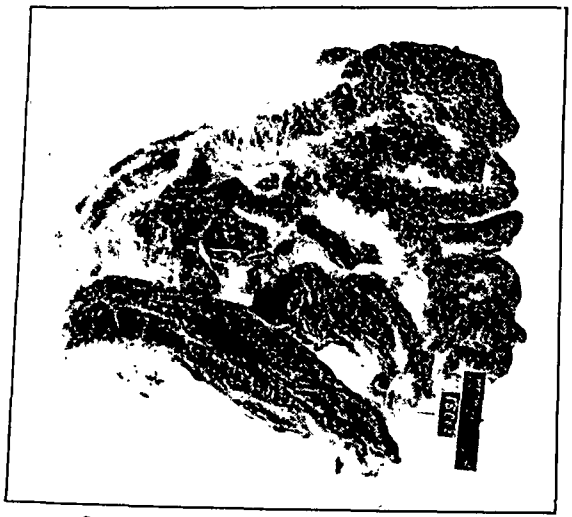

Fig. 8

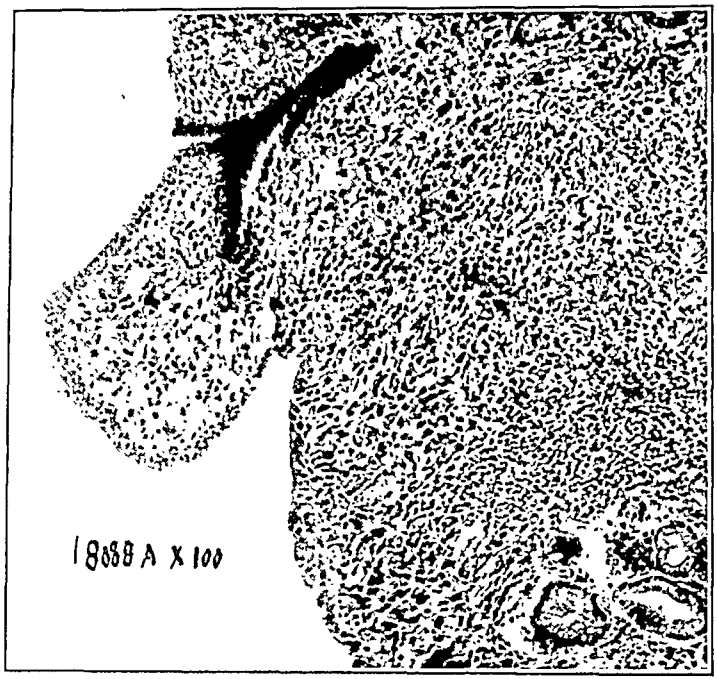

Fig. 7 (Case No. 18,088).—Stereogram of the pyloric one-third of the stomach, looking at the lesser curvature; multiple ulcers; a mass of cancerous tissue was removed from the area near the number needle.

Fig. 8 (Case No. 18,088).—Photomicrograph from the overhanging border of the ulcer. (× 100)

FIG. 9

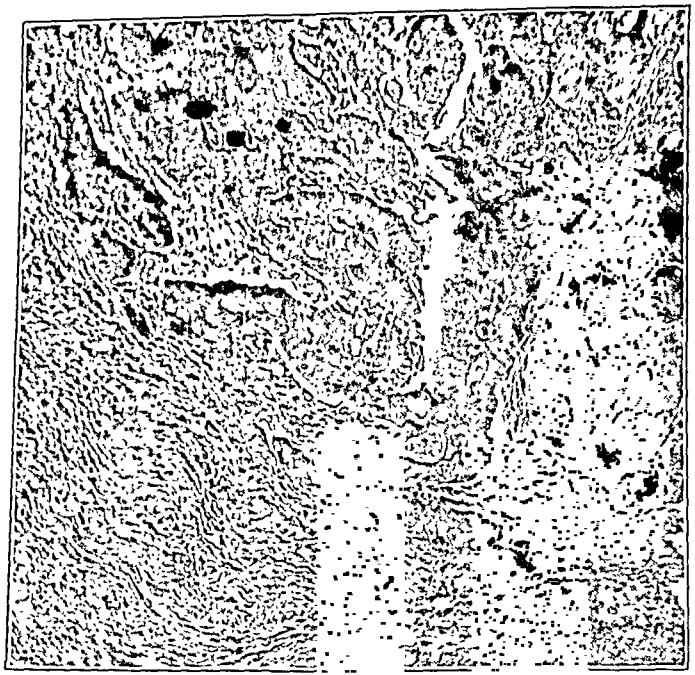

FIG. 10

FIG. 9 (Case No. 18,088).—Photomicrograph from an area deeper than that shown in Fig. 8. The epithelium shows aberrant proliferation and infiltration. (× 100)
FIG. 10 (Case No. 18,867).—Stereogram of the lesser curvature, showing proliferation of the muscularis by an ulcer with carcinoma in the border.

Fig. 11

Fig. 12

Fig. 11 (Case No. 18,867).—Photomicrograph of the base of the ulcer. (× 100)

Fig. 12 (Case No. 18,867).—Photomicrograph showing groups of epithelial cells partially cut off from the surface, actively proliferating but not infiltrating the surrounding tissues. (× 100)

FIG. 13

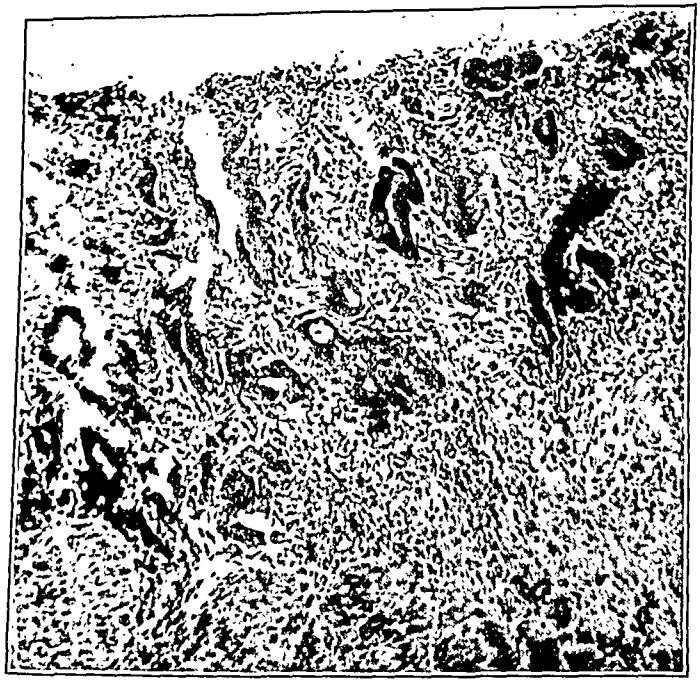

FIG. 14

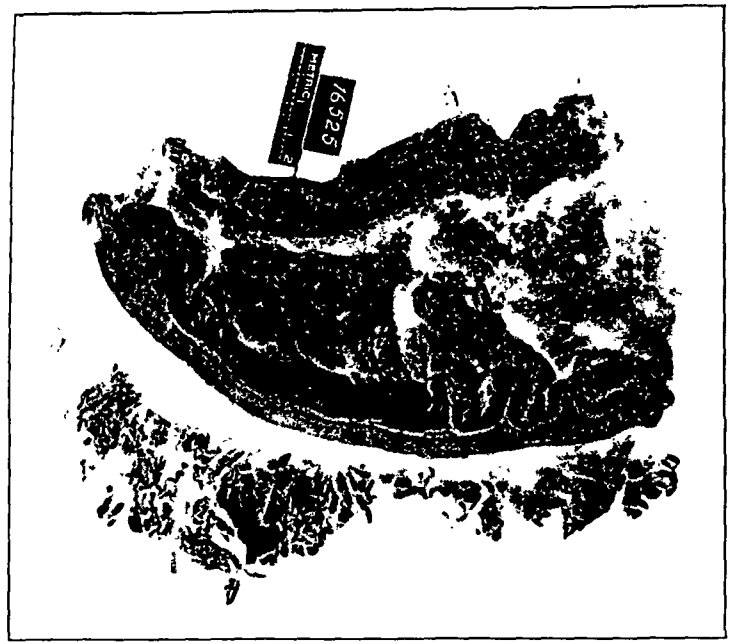

FIG. 13 (Case No. 18,867).—Photomicrograph showing typical carcinoma. (× 100)
FIG. 14 (Case No. 16,525).—Stereogram of the pyloric two-thirds of the stomach; carcinoma on a large ulcer beginning in the lesser curvature.

Fig. 15

Fig. 16

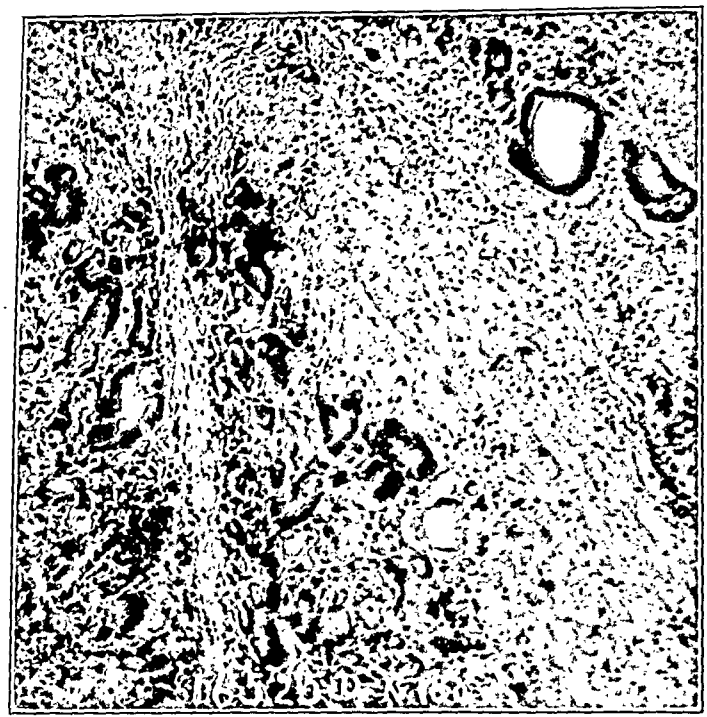

Fig. 15 (Case No. 16,525).—Photomicrograph showing a cross-section of the swollen glands with round-cell infiltration between the glands. (× 100)
Fig. 16 (Case No. 16,525).—Photomicrograph showing the bases of the glands clipped off by scar tissue. (× 100)

FIG. 17

FIG. 18

FIG. 17 (Case No. 16,525).—Photomicrograph showing active proliferation and much round-cell infiltration. (× 100)

FIG. 18 (Case No. 16,525).—Photomicrograph showing typical scirrhous cancer. Figs. 15–18 are from the border of the ulcer, but in successive microscopic steps away from its centre. (× 100)

FIG. 19

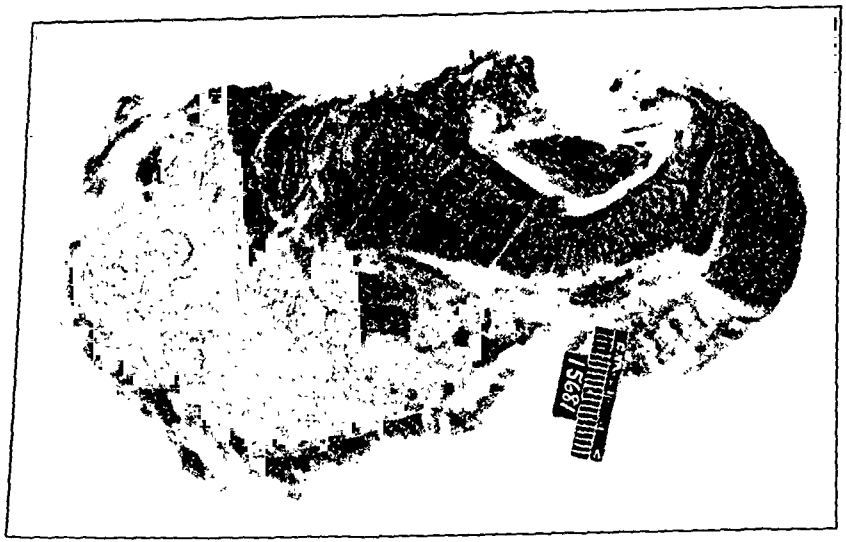

FIG. 20

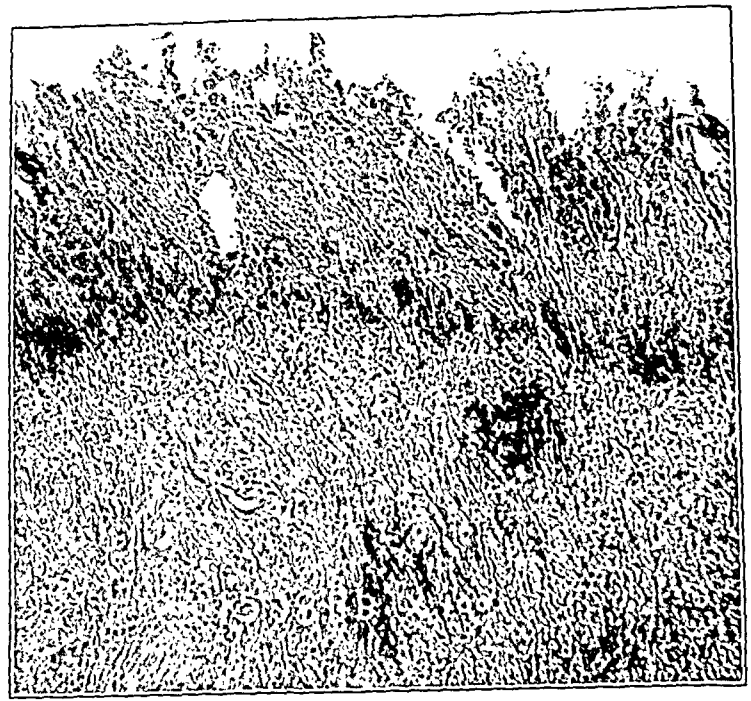

FIG. 19 (Case No. 15,681).—Stereogram of a portion of the pyloric one-half of the stomach, showing carcinoma of the lesser curvature involving also the pylorus.
FIG. 20 (Case No. 15,681).—Photomicrograph from the base of the ulcer. (× 100)

FIG. 21

FIG. 22

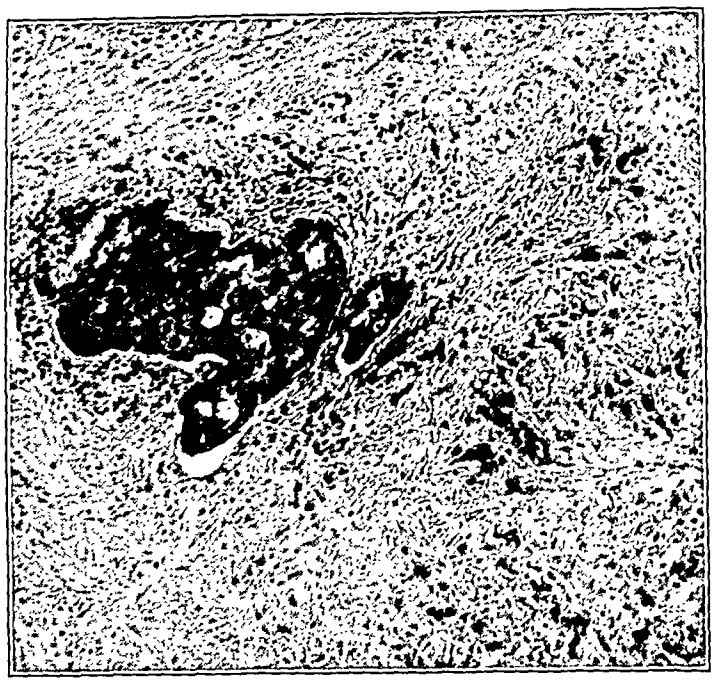

FIG. 21 (Case No. 15,681).—Photomicrograph showing proliferating epithelial masses at the base of the swollen mucosa; edge of the ulcer. (× 100)
FIG. 22 (Case No. 15,681).—Photomicrograph showing an area a little farther removed from the ulcerating area than the preceding section (Fig. 21). (× 100)

Fig. 23

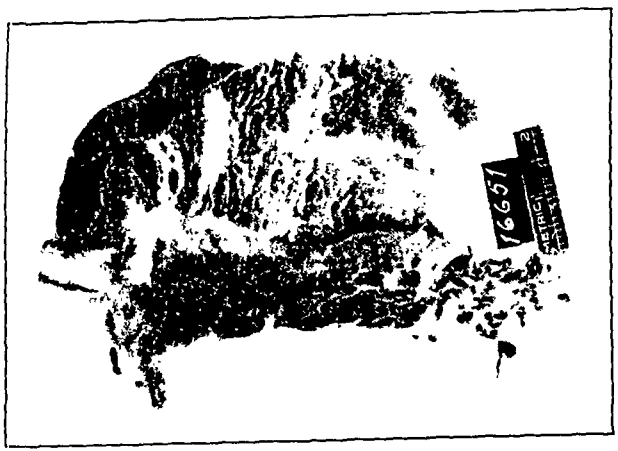

Fig. 24

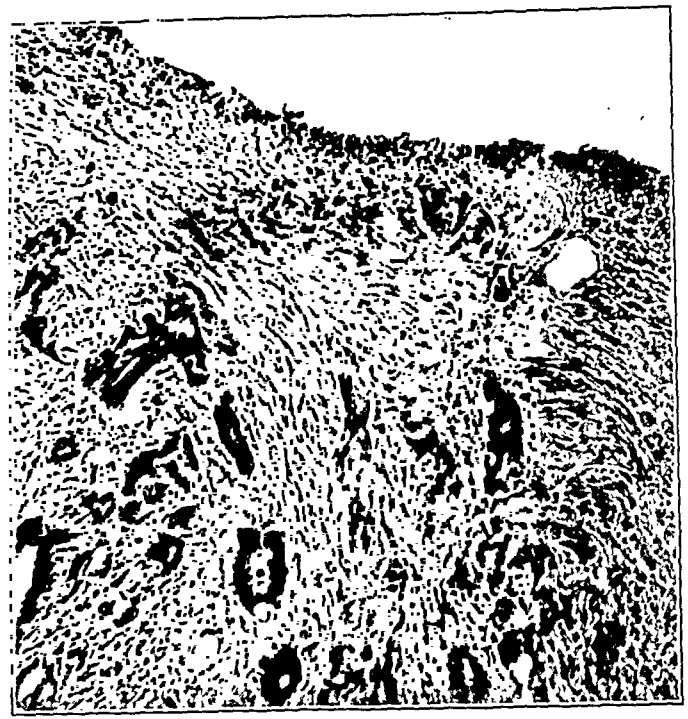

Fig. 23 (Case No. 16,651).—Stereogram of a portion of the pyloric one-third of the stomach showing carcinoma involving the lower lesser curvature and pylorus.
Fig. 24 (Case No. 16,651).—Photomicrograph of a section from the ulcer border, showing at the right side the ulcer base and at the left the developing carcinoma. (× 100)

Fig. 25

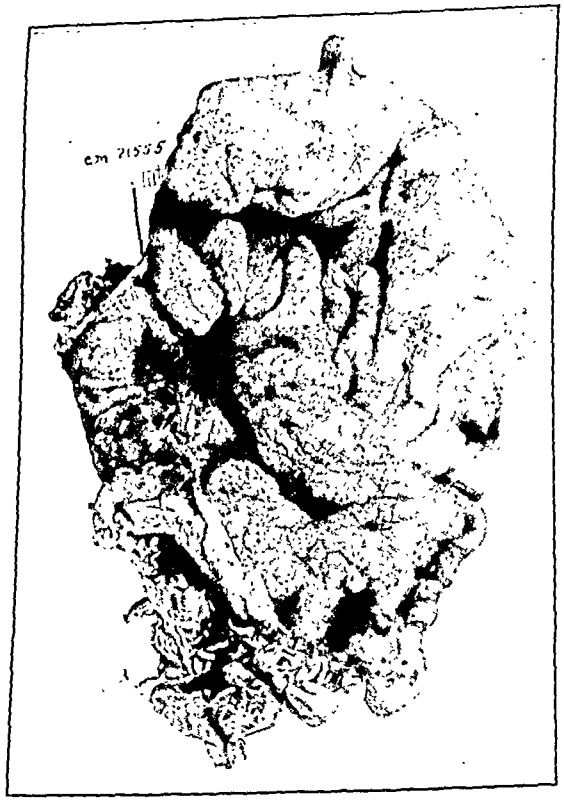

Fig. 26

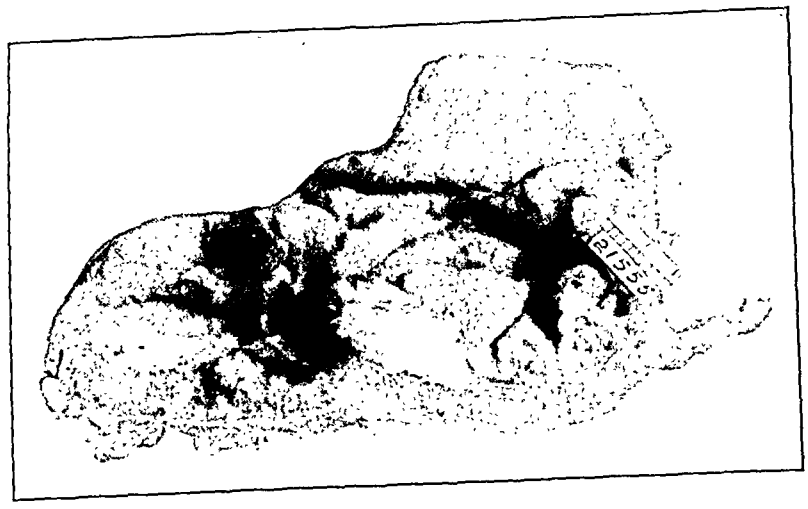

Fig. 25 (Case No. 21,555).—Stereogram showing carcinoma on a large ulcer of the lesser curvature of the stomach.
Fig. 26 (Case No. 21,555).—Stereogram of a gross section through a carcinoma and ulcer.

FIG. 27

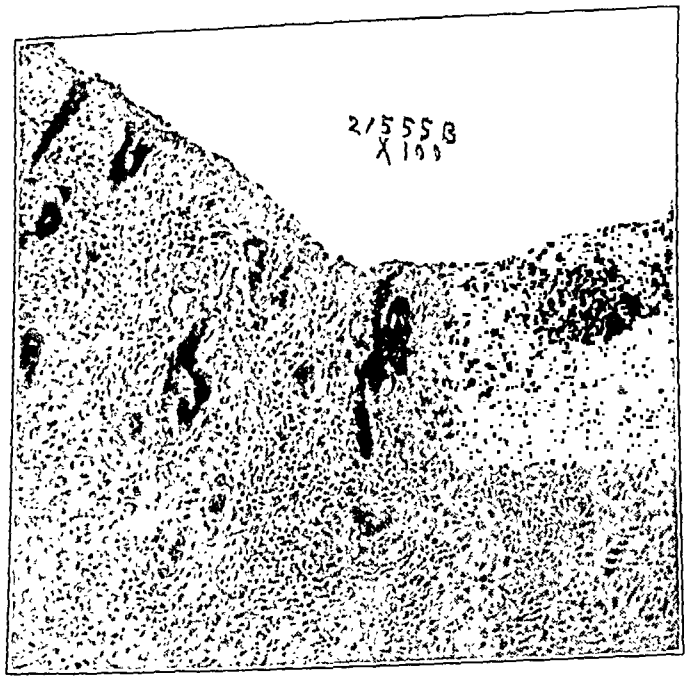

FIG. 28

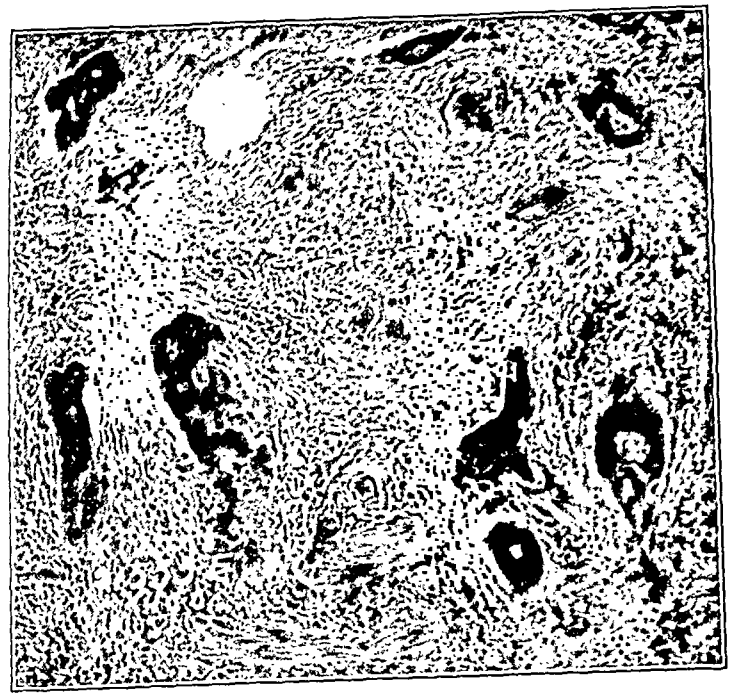

FIG. 27 (Case No. 21,555).—Photomicrograph from the edge of an ulcer; base of the ulcer at the right; separated bases of tubular glands showing early carcinomatous changes at the left. (× 100)

FIG. 28 (Case No. 21,555).—Photomicrograph of a section from under the base of the overhanging edge, showing scirrhous cancer. (× 100)

Fig. 29

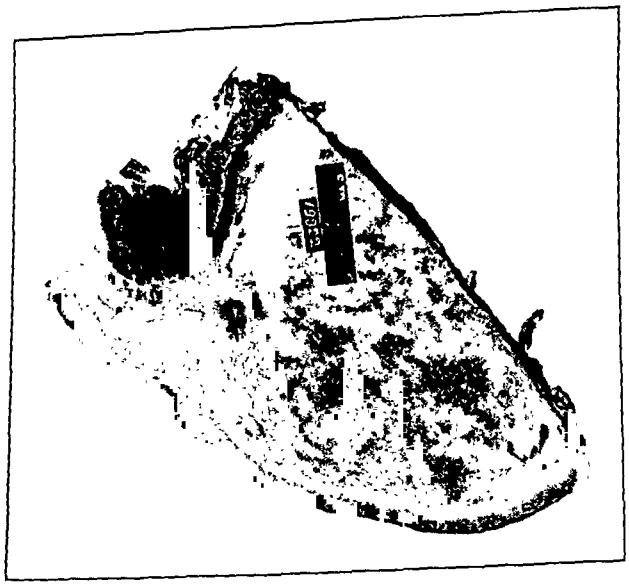

Fig. 30

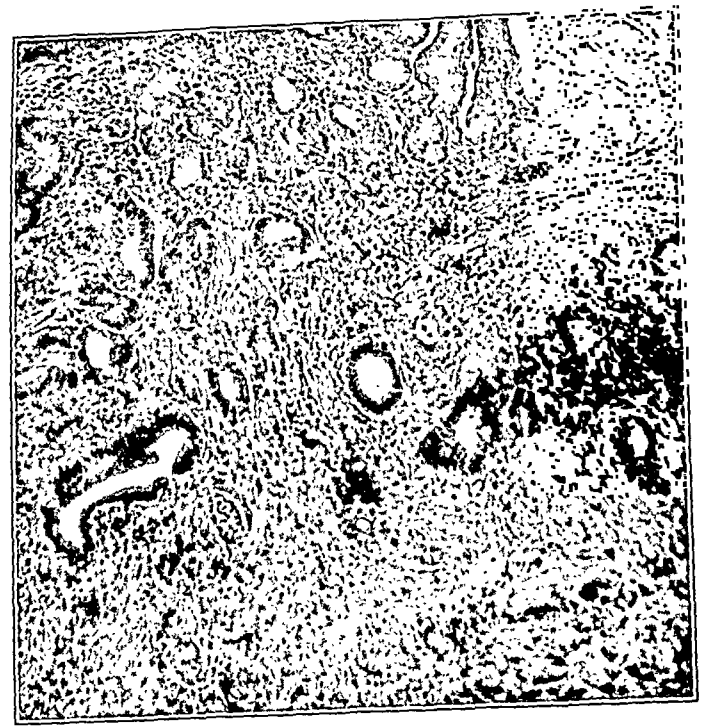

Fig. 29 (Case No. 19,322).—Stereogram showing a "ring cancer" of the pylorus (originated in an ulcer of the lesser curvature).
Fig. 30 (Case No. 19,322).—Photomicrograph showing islands of proliferating epithelium at the base of the mucosa of the overhanging border of an ulcer. (×100)

Fig. 31

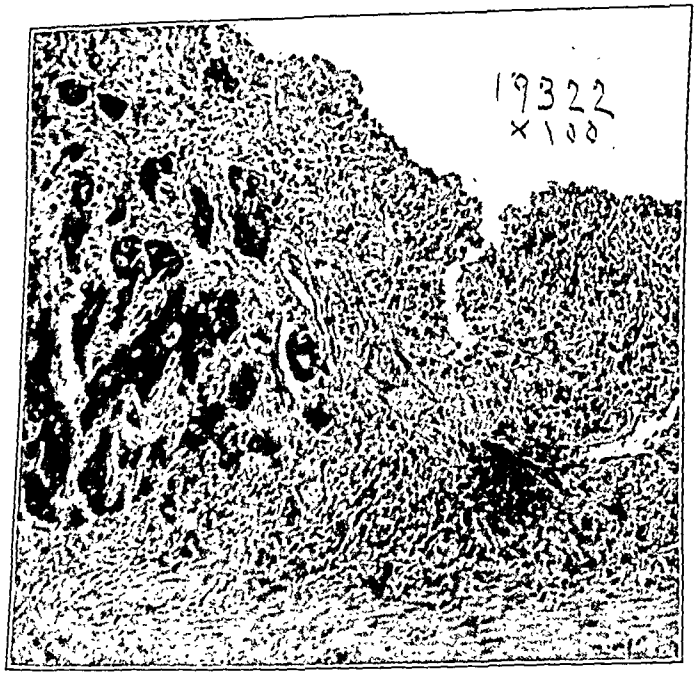

Fig. 32

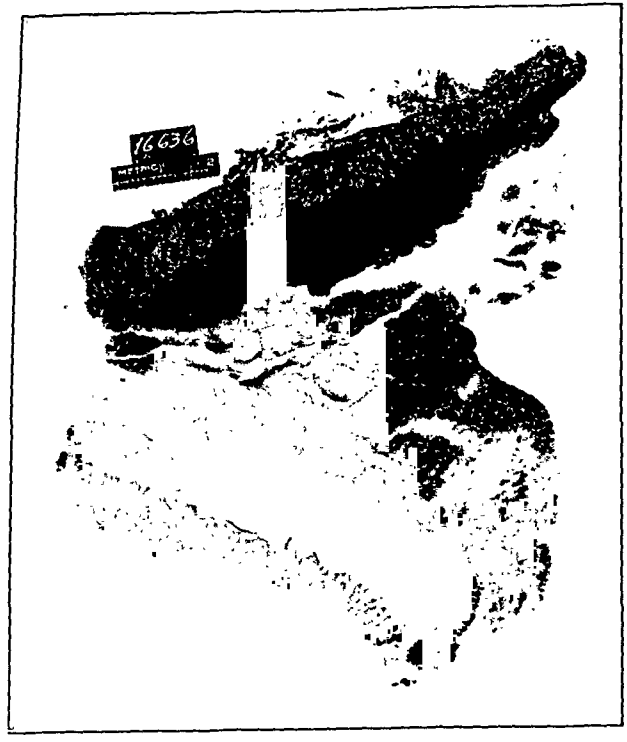

Fig. 31 (Case No. 19,322).—Photomicrograph showing the base of the ulcer at the right, and carcinoma in the border at the left. (× 100)
Fig. 32 (Case No. 16,636).—Stereogram showing a large ulcer of the lesser curvature with carcinoma in the border.

Fig. 33

Fig. 34

Fig. 33 (Case No. 16,636).—Photomicrograph showing a group of partially segregated tubular glands, and, at the lower right hand corner, one nest of aberrant proliferating epithelium. (× 100)

Fig. 34 (Case No. 16,636).—Photomicrograph showing the development of carcinoma in the scar tissue in the edge of an ulcer. (× 100)

FIG. 35

FIG. 36

FIG. 37

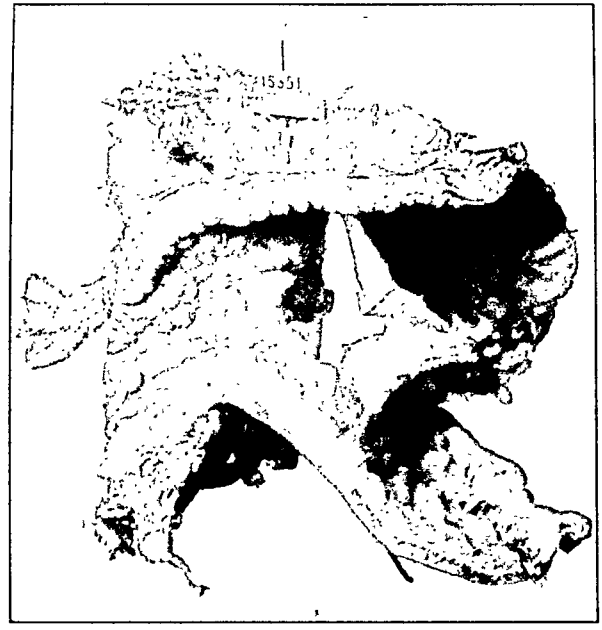

FIG. 35 (Case No. 14,857).—Stereogram showing a carcinoma filling the site of an old perforating ulcer of the stomach.
FIG. 36 (Case No. 14,857).—Photomicrograph showing a carcinoma with scar tissue. (× 100)
FIG. 37 (Case No. 15,351).—Stereogram showing a carcinoma in a small raised island of tissue in the base but near the edge of a large ulcer of the lesser curvature.

FIG. 38

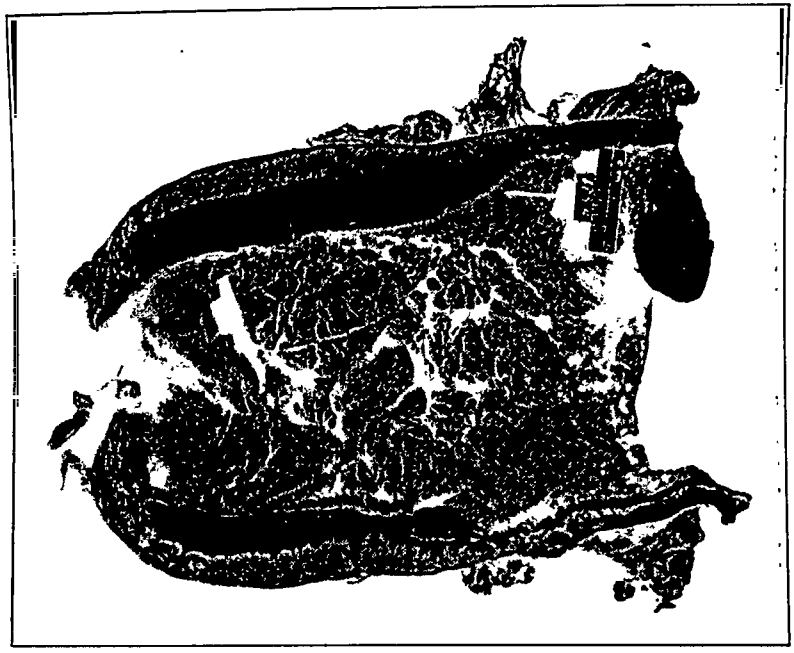

FIG. 39

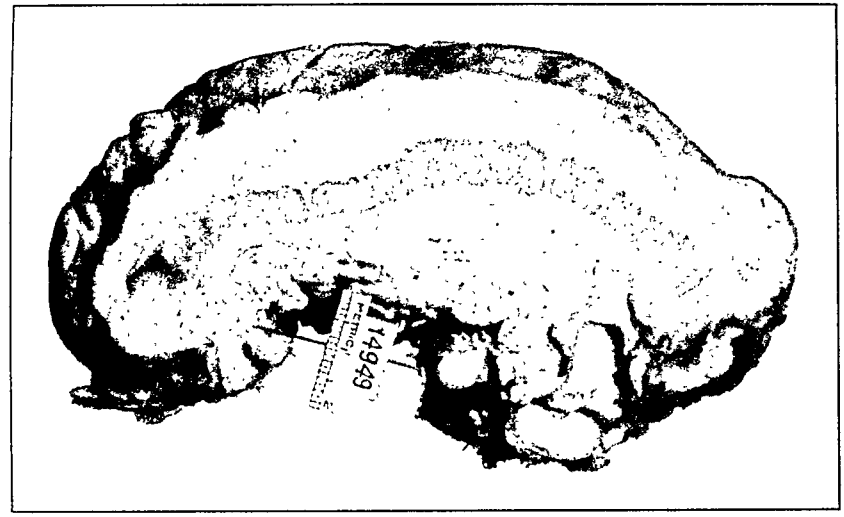

FIG. 38 (Case No. 18,514).—Stereogram showing an extensive carcinoma on a large ulcer of the lesser curvature.

FIG. 39 (Case No. 14,949).—Stereogram of a gross section through the pylorus and the lesser curvature showing an ulcer of the lower 8 cm. of the lesser curvature and carcinoma about 2 cm. above; metastasis in the glands.

FIG. 40

FIG. 41

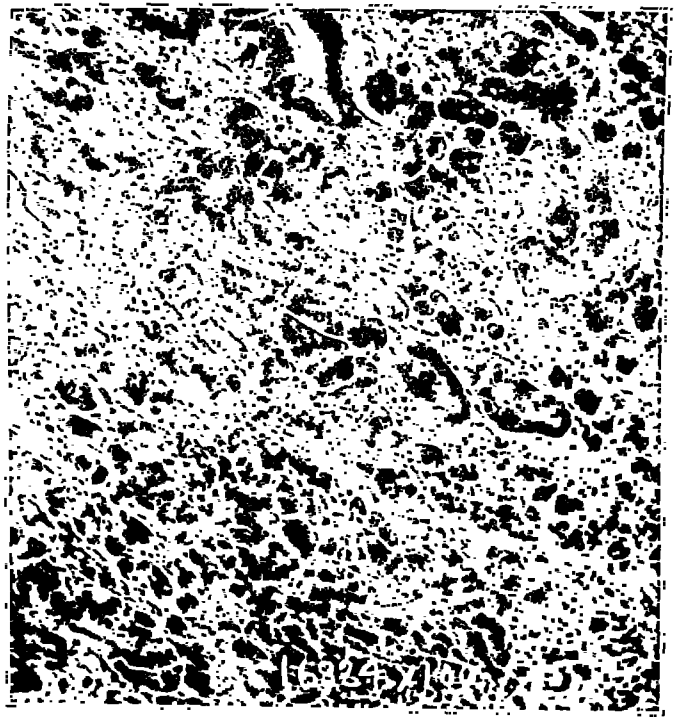

FIG. 42

FIG. 40 (Case No. 16,824).—Stereogram showing a small early carcinoma of the pylorus without pathological evidence of previous ulcer.
FIG. 41 (Case No. 16,824).—Photomicrograph showing an early adenocarcinoma. (× 100)
FIG. 42 (Case No. 16,806).—Stereogram of a portion of the stomach wall showing carcinoma with colloid degeneration and deposition of lime salts; no pathological evidence of previous ulcer.

involving also the pylorus. Fig. 20 is from a section from the base of the ulcer. Fig. 21 is of the overhanging border nearest the ulcerating area, showing segregated proliferating epithelial masses. Fig. 22 is of an area a little farther removed from the ulcerating area than the preceding section.

Case No. 16,651 (Fig. 23). This is a specimen from a woman, aged forty-two years, who for sixteen years had had intermittent stomach distress with vomiting, etc. For the last four months she had had considerable loss of strength, weight, and appetite. Stomach analysis showed a total acidity of 50, with free hydrochloric acid absent, lactic acid absent, and blood present. The gross specimen shows a lesion of the lesser curvature. Fig. 24 is of a section from the edge of the ulcer showing at the right side the old scar tissue in the border of the ulcer, and at the left side the carcinoma advancing into the scar.

Case No. 21,555 (Fig. 25). This specimen is from a man, aged sixty-seven years, who for twelve years had had considerable stomach distress, including vomiting, eructations, gas, etc. During the last few months he had grown considerably worse, had lost strength, appetite, and 35 pounds in weight. The stomach analysis showed a total acidity of 30, free hydrochloric acid 15, lactic acid absent, and blood present. The gross specimen showed a large ulcer of the lesser curvature, which is further shown in gross section in Fig. 26, in which the character of the ulcer can be more clearly made out. Fig. 27 shows the carcinoma advancing into the scar tissue at the edge of the ulcer. Fig. 28 is from material at the base of the overhanging mass, and shows scirrhous carcinoma change.

Case No. 19,322 (Fig. 29). This specimen is from a man aged thirty-three years, who for ten years had had symptoms of gastric ulcer, that is, distress, vomiting, gas, eructations, etc. During the last eight months he had had considerable loss of appetite with a loss of 50 pounds in weight. The stomach analysis showed a total acidity of 35, free hydrochloric acid 14, lactic acid absent, and blood absent. The gross specimen closely resembles a simple ulcerated pylorus. In one area, near the greater curvature, however, a nodular mass may be seen which suggests carcinoma. The character of the isolated islands of mucosa at this point is shown in Fig. 30; in Fig. 31 is shown the carcinomatous invasion of the scar tissue in the edge of the ulcer.

Case No. 16,636 (Fig. 32). This specimen is from a man, aged forty-six years, who for ten years had shown marked stomach symptoms, distress, gas, eructations, etc., and for the past eight months had loss of strength, appetite, and 70 pounds in weight. The stomach analysis showed a total acidity of 35, free hydrochloric acid absent, lactic acid present, and blood present. The specimen shows an enormous thickening of the muscularis and submucosa. On this mass of scar tissue is developing a carcinoma. Fig. 33 is of a

section from the lesser curvature area, showing a group of partially segregated tubular glands and near the lower edge of the figure a nest of aberrant proliferating epithelium. Fig. 34 shows the development of the carcinoma in the scar tissue.

Case No. 14,857 (Fig. 35). This specimen is from a man, aged fifty-one years, with a history of chronic stomach trouble for ten years, nausea, vomiting, gas, distress, etc. During the last six months these symptoms have been markedly increased and the patient has suffered a loss of 30 pounds in weight. The gross specimen shows the site of an old perforating ulcer, the muscularis being completely broken through, and the adhesions from the old perforation being quite evident. The carcinoma fills the site of the ulcer. Fig. 36 shows a carcinoma developing within the scar tissue. Judging from the microscopic appearance alone, one might hesitate to decide that this was a case of precedent ulcer, although the bands of scar tissue with masses of epithelium included are quite suggestive. The gross specimen with the history, however, is sufficient to warrant a positive diagnosis.

Case No. 15,351 (Fig. 37). This specimen is from a man, aged forty-one years, who for many years (fifteen or more) had had gastric distress, vomiting, and eructations of gas. In the last six months he had had a loss of strength, and a loss in weight of 30 pounds. The stomach analysis showed a total acidity of 25, free hydrochloric acid absent, lactic acid present, and blood present. The gross specimen shows a large ulcer of the lesser curvature and a small carcinoma developing in a raised island of tissue in the base of the ulcer near one edge. It is the only instance of the kind in our series.[4]

Case No. 18,514 (Fig. 38). This specimen is from a female, aged forty-one years, who had for three years suffered gastric distress, vomiting, gas, eructations, etc. In the last six months she had had marked loss of appetite, loss of strength, and loss in weight (60 pounds). The stomach analysis showed a total acidity of 100, free hydrochloric acid absent, lactic acid present in large amounts, and blood present. The specimen showed a widely diffused cancer on a large ulcer of the lesser curvature.

Case No. 14,949 (Fig. 39). This specimen is from a man, aged thirty-one years, from whom no history of any stomach trouble could be elicited prior to that beginning one year ago, when he began to have nausea, vomiting, loss of strength, and loss of 65 pounds in weight. Stomach analysis showed a total acidity of 10, free hydrochloric acid absent, lactic acid present, and blood present. A gross section through the lesser curvature of the stomach is here shown. The stomach wall at this point was from 2.5 to 4 cm. thick. The lesion was purely of an ulcerous character for the lower 8 cm. of the

[4] The microscopic details in this and most of the succeeding cases are omitted for lack of space. They were closely parallel with those already shown.

lesser curvature; about 2 cm. of the upper portion of the lesser curvature showed a rapidly growing carcinoma, which had formed metastases in the adjacent glands.

This case is presented to show the unreliability of ever so well taken clinical histories, particularly in the young male, who is not accustomed to give much attention to slight stomach trouble. There can be no question that this patient had stomach ulcer for years preceding the onset of cancer.

The preceding nine cases are fair representatives of those gastric resections for carcinoma in which we consider the pathological evidence of preceding ulcer sufficient to warrant such a diagnosis.

Case No. 16,824 (Fig. 40). This specimen is from a man, aged forty-three years, who was apparently well until one year ago, when he began to show stomach distress, with gas, loss of strength, and loss of weight (40 pounds). The stomach analysis showed a total acidity of 25, with free hydrochloric acid 8, lactic acid present, and blood present. The specimen shows a small carcinoma of the pylorus, without gross evidence of previous ulcer. Fig. 41 is from a deep level of a section and shows adenocarcinoma. We would not seem to be warranted in making any diagnosis of preceding ulcer in this case of carcinoma, either from the history or the pathological evidence.

Case No. 16,806 (Fig. 42). This is a specimen from a man, aged sixty-eight years, who during the last thirty years had had three prolonged attacks of stomach trouble, marked distress, gas, vomiting, eructations, etc. During the last eight months he had had loss of strength, loss of appetite, and a loss of 30 pounds in weight, and there was also severe persistent pain. Stomach analysis showed a total acidity of 30, free hydrochloric acid absent, lactic acid present, and blood present. Two-thirds of the stomach was removed. Only a small portion of the specimen is here shown—a section through the wall of the lesser curvature, which was about 3 cm. thick. Colloid degeneration with deposition of lime salts had occurred throughout the wall of the removed portion of the stomach. In this case there is absolutely no pathological evidence of the occurrence of previous ulcer, although the thirty-year history is very strong clinical evidence.

These last two cases represent the group of 33 (22 per cent.) of our cases, in which we could find insufficient pathological evidence to warrant a diagnosis of preceding ulcer. The latter case also is a fair example of the type of case which frequently comes to autopsy and shows no evidence of preceding ulcer.

The preceding cases are fair representatives of our series. The 109 cases (71 per cent.) which present pathological evidence, gross and microscopic, parallel with that shown in detail herewith, that is, large ulcers with scar tissue centres and overhanging borders, deep in the bases of which cancer is present, in almost every instance have unmistakably originated on the lesser curvature of the stomach, the

usual site of gastric ulcer. Further, almost every case gives a clinical history suggesting gastric ulcer for a long period of years preceding the relatively short period when the history became that of gastric cancer.

That carcinomas should develop in the edges of gastric ulcers is only what we should expect; the wonder is that the facts should have been so long in being recognized. This has been due to: (*a*) Failure to recognize clinically the frequency of gastric ulcer; (*b*) failure to recognize that gastric cancers are not initially pyloric tumors, but extensions thereto from the lesser curvature; and (*c*) giving undue weight to observations at autopsies. When the patient has died of gastric cancer, the neoplasm has usually obliterated all gross and microscopic evidence of previous ulcer.

As the pathologist examines stomach specimens from the surgical clinic he constantly observes the various steps in the following sequence:

1. Chronic ulcers from the centres of which the mucosa has disappeared leaving a scar tissue base.
2. In the overhanging broders of the ulcers the mucosa is proliferating.
3. Deep in the borders many groups of epithelial cells have been nipped off by scar tissue and are exhibiting all stages of aberrant proliferation with infiltration of the surrounding tissues.
4. Metastases are forming in the lymphatics of the stomach wall and adnexa.

A small percentage of cases operated upon are too far advanced to show these steps, and a very small percentage—probably not over 2 per cent.—give evidence of rapid aberrant epithelial proliferation and infiltration without any sign of previous ulcer.

Adopting Adami's[5] classification we may therefore correctly desig-designate most gastric carcinomas as "blastomas originating from unipotential cells of postnatal displacement," although it is probable that a very small number are "blastomas originating from unipotential cells that assume neoplastic characters without displacement and rapidly assume malignancy."

[5] Principles of Pathology, 1908 i, 770.

THE ETIOLOGY AND PATHOLOGY OF INGUINAL HERNIA.

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In order to understand the mode of occurrence of inguinal hernia and employ intelligent operative measures for its cure, the surgeon must be familiar with those structural conditions which make it possible for a portion of the intra-abdominal contents to protrude through the inguinal triangle, a space bounded above by a line drawn horizontally inward from the junction of the middle and outer thirds of Poupart's ligament to the outer margin of the rectus muscle, below by the inner two-thirds of Poupart's ligament, and internally by the outer margin of the rectus muscle. The structures to be considered are the inguinal fossæ, Hesselbach's triangle, the internal abdominal ring, the inguinal canal, and the external abdominal ring.

When the anterior abdominal wall is viewed from behind the following structures are seen: Extending from the apex of the bladder to the umbilicus is the urachus, the degenerated intra-embryonic portion of the allantois, also known as the median vesico-umbilical ligament. On either side of this median cord there is another, extending obliquely upward and inward to join it at the umbilicus. These are the remains of the hypogastric arteries, which are patent only during intra-uterine life. They are also called the lateral vesico-umbilical ligaments. Lying external to these lateral cords are the deep epigastric arteries. These five structures are covered with peritoneum, arranged in more or less well-defined folds, that covering each obliterated hypogastric and deep epigastric artery being known as the plica hypogastrica and plica epigastrica respectively.

In relation with these cords three depressions may be distinguished—one between the urachus and the obliterated hypogastric artery, another between the hypogastric and the epigastric arteries, and a third external to the epigastric artery. These depressions are known as the internal, middle, and external inguinal fossæ respectively, and with the exception of the internal one are very important with reference to the occurrence of inguinal hernia (Fig. 1). They can be examined better after the peritoneum has been carefully stripped off. When this has been done it will be found that the internal fossa is bounded in front, with the exception of a small area above its inferior external angle, by the rectus abdominis muscle, which is strong and unyielding, and well adapted

to withstand any unusual intra-abdominal pressure which may be exerted upon it. Therefore, only the small area of this space which is not covered by the rectus muscle can be considered as a weak spot in the abdominal wall. It is quite different, however, with the middle fossa, the one between the obliterated hypogastric and the deep epigastric arteries. This one is bounded in front only by the transversalis fascia and the attenuated lower fibers of

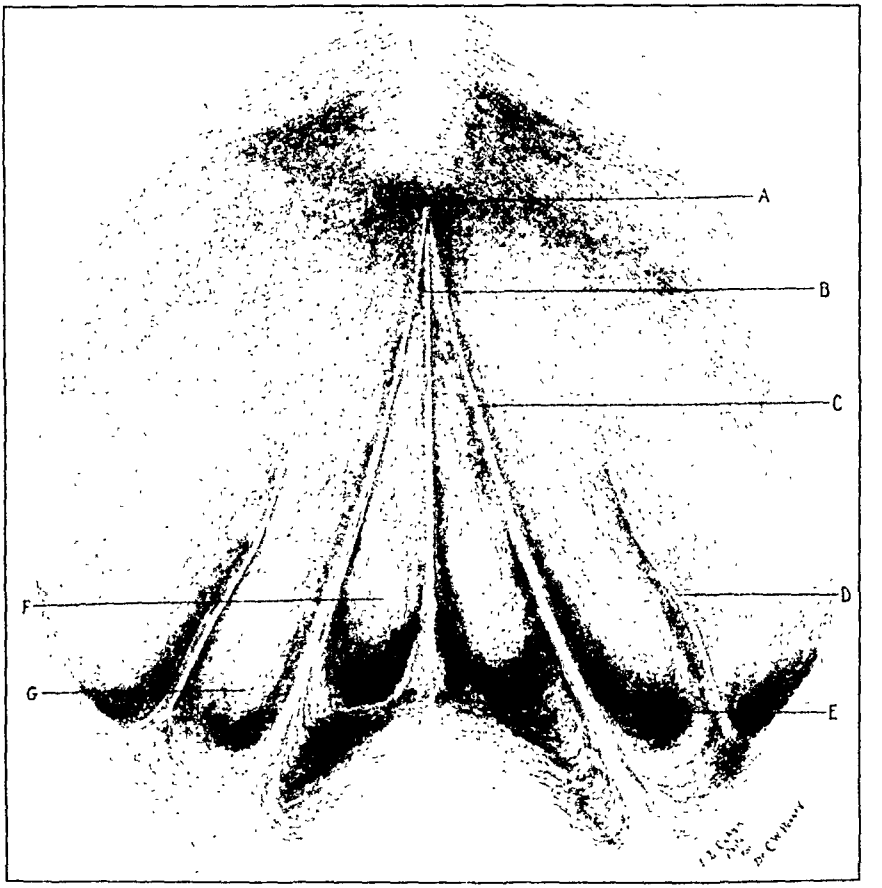

FIG. 1.—The anterior abdominal wall viewed from behind, showing the inguinal fossæ: *A*, umbilicus; *B*, urachus; *C*, obliterated hypogastric artery; *D*, deep epigastric artery; *E*, external inguinal fossa; *F*, internal inguinal fossa; *G*, middle inguinal fossa. (Drawing made from a dissection prepared by Dr. Bonney, Department of General Anatomy, Jefferson Medical College.)

the internal oblique muscle. An examination of several hundred cadavers has shown that there is not a decided blending of the lower fibers of the internal oblique muscle with those of the transversalis to form a well-developed conjoined tendon in more than one-fourth of all cases. That portion of the internal oblique arising from Poupart's ligament has often been found to be aponeurotic instead of muscular, and to fuse almost imperceptibly with the

aponeurosis of the transversalis; whether it stops short or is continued downward with the latter to help form the deep crural arch cannot positively be stated, for the reason that it has not been possible satisfactorily to dissect the one from the other so homogeneously are they united. Even when a decided tendinous union takes place there is always a considerable area below it which is filled in only by fascia. Thus, it is seen that the middle fossa is a decidedly weak area in the anterior abdominal wall, ill adapted to resist the force of any unusual intra-abdominal pressure which may be brought to bear upon it. In fact, it is through this area and the contiguous small uncovered portion of the internal inguinal fossa already described that a certain percentage of inguinal herniæ occur. The space thus formed, which is bounded externally by the deep epigastric artery, internally by the outer margin of the rectus muscle, and inferiorly by Poupart's ligament, is known as Hesselbach's triangle.

The external inguinal fossa, lying external to the deep epigastric artery, constitutes another weak spot in the anterior abdominal wall. This fossa really owes its existence to the internal abdominal ring, a spot in the transversalis fascia through which the testicle pushes its way on its course downward from the abdominal cavity to the scrotum. If the transversalis fascia be examined carefully at this spot after the peritoneum has been stripped away, the following arrangement will be found. Internally it is strengthened by tendinous fibers passing upward from the inner end of Poupart's ligament, and externally and superiorly it presents a somewhat annular arrangement of its fibers, which extend downward along the spermatic cord. To this funnel-shaped process, the term infundibuliform fascia is applied, and the internal stronger margin is known as the interfoveolar ligament of Hesselbach. The latter really forms a line of dermarcation between the external and middle fossæ just as the deep epigastric artery does (Fig. 2).

The formation of the internal ring and the inguinal canal is best made clear by tracing the descent of the testicle from the abdomen to the scrotum. The scrotum is formed from the genital swellings, into which a little sac of peritoneum is invaginated comparatively early in fœtal life. The inguinal ligaments, or gubernacula, are also attached to the bottom of this peritoneal pouch. Now as the fœtus develops, this pouch (which in the male is called the vaginal process and in the female the canal of Nuck) becomes more and more elongated, keeping pace with the growth of the genital swellings, until toward the termination of intra-uterine life, probably at about the end of the eighth month, it has formed a sac extending well down into the scrotum. It is to be borne in mind that this sac is placed in front of the testicle before that organ emerges from the abdomen, that it always bears such a relation to the testicle, and that it is not pushed from the anterior abdominal

wall by the testicle in the way in which the other layers enveloping that gland and the spermatic cord are displaced. From these considerations it becomes evident that at a certain stage of fœtal life there is a free communication between the peritoneal cavity and the scrotum. Shortly after birth—probably between the tenth and twentieth days—the vaginal process normally becomes obliterated except as to its lower portion, which surrounds the testicle

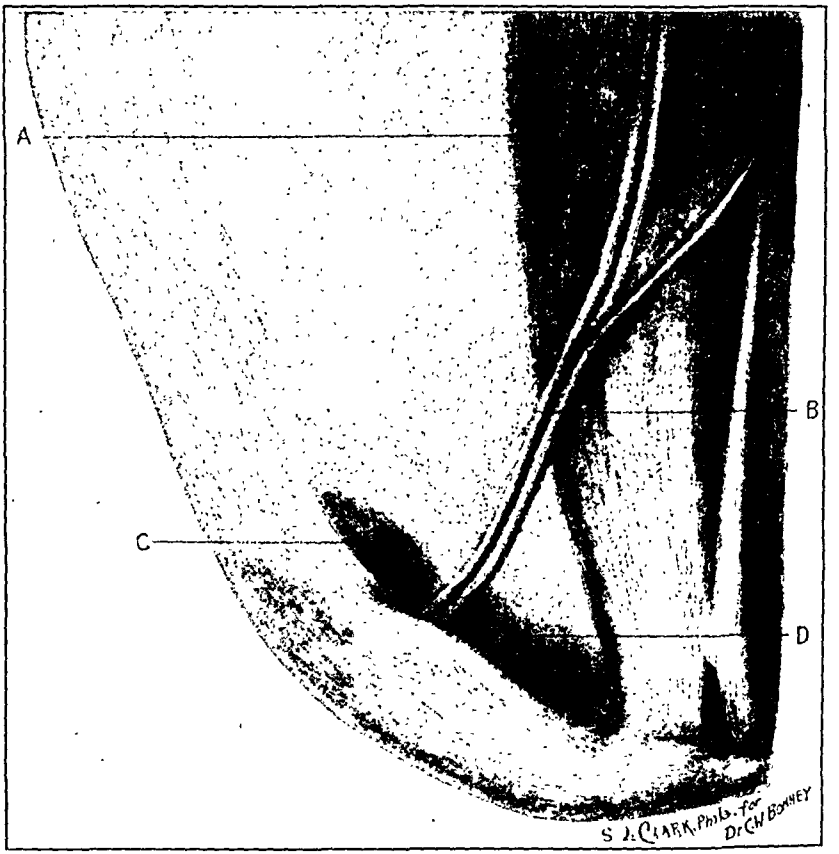

FIG. 2.—The anterior abdominal wall viewed from behind after the peritoneum has been stripped up: *A*, rectus muscle; *B*, deep epigastric vessels; *C*, external inguinal fossa; *D*, middle inguinal fossa. Observe that the internal inguinal fossa is almost entirely covered by the lower portion of the rectus. (Drawing made from a dissection prepared by Dr. Bonney, Department of General Anatomy, Jefferson Medical College.)

and forms its tunica vaginalis. Failure of the upper portion of this peritoneal process to become obliterated constitutes an important factor in the production of congenital hernia. Reference to it will be made again.

By what force or forces the testicle is made to change its position from abdomen to scrotum has been a subject of considerable discussion. In the light of our present knowledge it may be stated that the conditions operative in bringing about this change are

the disparity of growth between the inguinal ligaments and the abdominal wall and the elongation of the vaginal process of peritoneum. Modern embryologists are agreed that the former factor is a very important one. As the abdominal walls develop more rapidly than the inguinal ligaments, there is a tendency for the testicles to be drawn down toward the inguinal region where the ligaments are attached. As this attachment is into the little sac of peritoneum in the genital swellings which develops into the vaginal process, it seems reasonable to suppose that the testicle will be carried still further downward as the process becomes elongated. The theory that the ligament itself draws the testicle downward by contracting is not tenable in view of our present embryological knowledge.

As the testicle passes through the abdominal wall it carries in front of it a layer from each of the muscular or fascial planes which it perforates. Thus, as it comes out of the abdomen at the spot known as the internal ring, it carries a portion of transversalis fascia along, drawing it down like a cone or funnel. Hence the application of the term infundibuliform fascia to this portion of the transversalis fascia. From the next stratum of the belly wall, the internal oblique muscle, it takes up a covering known as the cremaster, and as it passes through the succeeding layer, the aponeurosis of the external oblique muscle, it pushes a fascial investment called the external spermatic fascia in front of it.

The spermatic cord occupies and fills up the channel thus made by the testicle in the substance of the abdominal wall. This channel is known as the inguinal canal. It extends from the internal to the external abdominal ring, and in the adult is about an inch and a half long. It is bounded anteriorly by the aponeurosis of the external oblique muscle and in its outer third by the internal oblique, posteriorly by the transversalia fascia, superiorly by the arched fibers of the internal oblique and transversalis muscles, and inferiorly by Poupart's ligament. A study of hundreds of dissections has convinced us that these structures form the true boundaries of the canal, and that any additional ones which may be given are artificial, inaccurate, and confusing.

It must always be borne in mind that the term "canal" is a misnomer. There was a canal in fœtal life, but under normal conditions there is none in extra-uterine life except for a very short time. In the adult the inguinal canal is nothing but a slight gap in the abdominal wall well filled in by the spermatic cord or round ligament. From the description of the internal abdominal ring it will be readily understood that the term "ring" is also inaccurate (Fig. 3).

The external abdominal ring likewise is not a ring in the true sense of the word. It is a cleft in the aponeurosis of the external oblique muscle, situated just above and external to the spine of the

pubes, having its base below and its apex above. It transmits the spermatic cord in the male and the round ligament in the female. The margins of this cleft are called pillars, of which the outer is the stronger, being formed by the inner end of Poupart's ligament. The inner pillar is attached to the symphysis pubis, its fibers interdigitating with those of the internal pillar on the opposite side. Stretching across the ring from one pillar to the other are fibers likewise derived from the aponeurosis of the external oblique, known as the intercolumnar fascia, and continuous with the external spermatic fascia which envelopes the cord. Thus it is seen that there is no distinct opening in the aponeurosis of the external oblique muscle, the cord and intercolumnar fascia effectually closing in the breach between its fasciculi. It is only after the intercolumnar fascia has been removed and the cord displaced that the gap becomes apparent (Figs. 4 and 5).

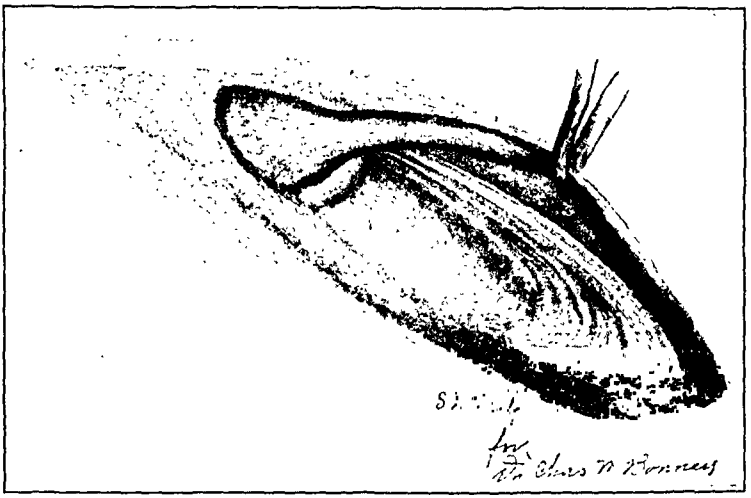

FIG. 3.—The inguinal canal and the internal abdominal ring. The spermatic cord has been lifted out of the canal, and traction is being made upon it so as to render the infundibuliform fascia tense. Observe the deep epigastric artery internal to the ring. (Drawing made from a dissection prepared by Dr. Bonney, Department of General Anatomy, Jefferson Medical College.)

As has previously been stated, a certain number of all inguinal herniæ, certainly not more than four or five out of every hundred, protrude from the abdomen through Hesselbach's triangle. The remainder come out through the internal abdominal ring. They traverse the inguinal canal, break through the external ring, and either present above and external to the pubes or pass downward into the scrotum. A hernia of this kind is known as an indirect, oblique, or external hernia: indirect, in contradistinction to the one which comes out through Hesselbach's triangle; oblique, because of the direction in which it passes downward; external, because it lies to the outer side of the deep epigastric artery. A hernia

which passes through the external ring is called a complete inguinal hernia. The coverings of an indirect hernia are derived from the various layers of the abdominal parietes, just as are the coverings of the spermatic cord. From within outward they are peritoneum, preperitoneal fat, infundibuliform fascia, cremaster muscle or fascia, intercolumnar fascia, superficial fascia, and skin. Two points in reference to these coverings are worthy of mention: (1) The peritoneal covering, or sac, of an acquired hernia is derived from the parietal peritoneum, so that when the hernia extends down into the scrotum, its peritoneal covering has nothing in com-

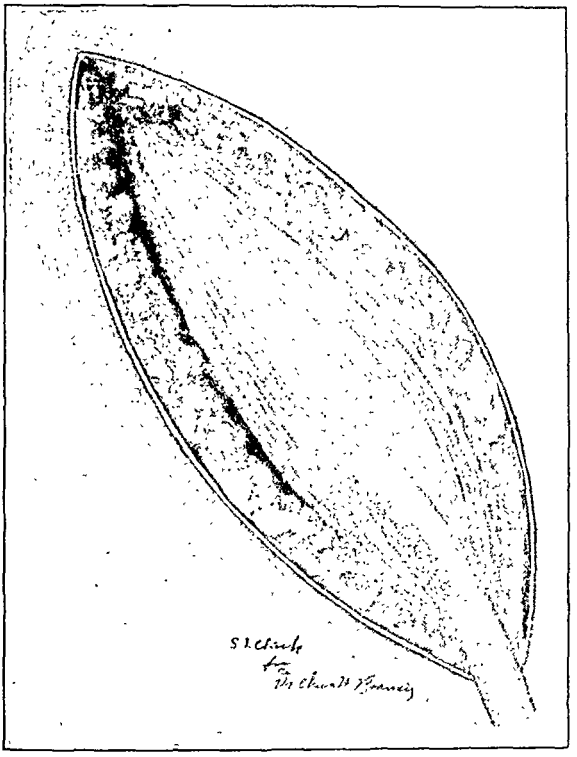

FIG. 4.—Showing the spermatic cord emerging from the external abdominal ring. Observe that there is no distinct opening in the aponeurosis of the external oblique muscle. (Drawing made from a dissection prepared by Dr. Bonney, Department of General Anatomy, Jefferson Medical College.)

mon with the tunica vaginalis testis. This is not true, however, of congenital herniæ, presently to be considered. (2) In enumerating the coverings of a scrotal hernia, the term dartos may be substituted for superficial fascia, the dartos being continuous with the superficial fascia in the lower third of the abdomen, but containing some muscle fibers. Although this point may seem trivial it has often been puzzling to students. Possibly this may be the most appropriate place to state that a scrotal hernia is merely a complete inguinal hernia, either indirect or direct, which has reached the scrotum.

A hernia coming through Hesselbach's triangle is called a direct or internal hernia: direct, for the reason that it bulges forward into the lower part of the inguinal canal without following the course of the cord obliquely downward from the internal ring; internal, because it lies to the inner side of the deep epigastric artery. As has already been stated, such a hernia almost always comes out through the middle inguinal fossa. We cannot agree with those writers who state that it more commonly emerges from the internal fossa. The latter is well protected by the rectus muscle in the manner previously described. That a loop of gut may work its

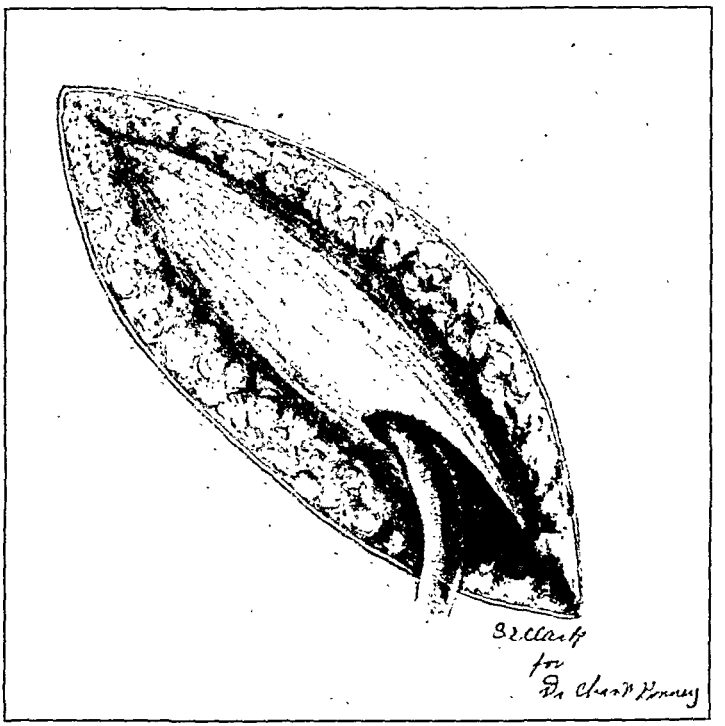

FIG. 5.—Showing the external abdominal ring after the intercolumnar fascia has been removed and the spermatic cord freed. Observe the opening in the aponeurosis of the externa oblique muscle. (Drawing made from a dissection prepared by Dr. Bonney, Department of General Anatomy, Jefferson Medical College.)

way through the unprotected lower external corner of this fossa is not to be doubted, but it is much more likely to come out through the middle fossa, both lateral boundaries of which are less resistant than the strong tough margin of the rectus muscle.

A hernia escaping through the internal fossa has been termed an internal oblique or vesicopubic hernia. When the sac attains a considerable size it would be difficult to trace its origin unless the urachus could be readily distinguished. It is interesting to note that isolated cases are on record in which a hernia has separated

the fibers of the rectus muscle, and even pushed its way between the two recti, presenting at the linea alba.

The coverings of a direct hernia are, from within outward, peritoneum, preperitoneal fat, transversalis fascia, intercolumnar fascia, superficial fascia, and skin. The conjoined tendon is usually enumerated among the coverings of a direct hernia, but, as already stated, it is only in about 25 per cent. of all subjects that such a structure is actually present. When it does exist it is not pushed forward by the hernia, as has generally been taught, but the hernia slips around it, leaving it undisturbed. The tendon, when present, is firmly fixed to the crest of the pubes and iliopectineal line, and above is continuous with the substance of the internal oblique and transversalis muscles. That it could be pushed forward by a hernia is not at all probable, and, moreover, we have never found it displaced in those cases which we have observed at the operating table. In those cases in which there is no conjoined tendon it is possible that some of the attenuated lower fibers of the internal oblique may be carried forward by the hernia, especially if the latter is large. In about forty operations for direct hernia, however, one of us has never observed any muscle fibers in the coverings of the bowel.

It has been frequently stated that about one-fifth of all inguinal herniæ are direct. We are of the opinion that this estimate is much too high. We believe, as we have already stated, that not more than four or five out of every hundred are direct.

Etiology. The causes of inguinal hernia may be considered under three headings: (1) Congenital defects; (2) natural weakness of the abdominal wall in the inguinal triangle, and forces which increase this weakness; and (3) conditions which increase intra-abdominal pressure.

Congenital defects relate chiefly to faulty obliteration of the vaginal process of peritoneum. As previously stated, that portion extending from the internal ring to the top of the testicle usually becomes obliterated between the tenth and twentieth days after birth. The exact manner in which closure takes place is not known. It is generally conceded that obliteration begins in the middle part of the process, and some who have studied the subject also think it commences simultaneously near the internal ring. At all events there are a certain number of individuals in whom closure partly or entirely fails to take place. These persons are particularly predisposed to hernia. When the vaginal process remains open throughout its whole extent there is an uninterrupted passage from the peritoneal cavity to the scrotum into which the intestine can readily find its way, with the result that a hernia is produced. Such a hernia is called congenital, its sac being preformed. The contents of the sac are in contact with the testicle, but the testicle is not suspended by its cord within the sac in the manner that a

chandelier is suspended from the ceiling into the middle of a room, although we have found that undergraduates and practitioners of medicine alike sometimes entertain this erroneous conception of the relation of the one to the other. The inguinal portion of the cord lies in the retroperitoneal connective tissue, the scrotal portion is also posterior to the vaginal process, and one layer of peritoneum covers the testicle, namely, the visceral layer of the tunica vaginalis, which is identical with the posterior wall of the hernial sac. Thus, the testicle really lies behind the sac, being separated from the gut by a single layer of peritoneum instead of a double one, as is the case with acquired indirect inguinal hernia. These relations are well illustrated in Fig. 6.

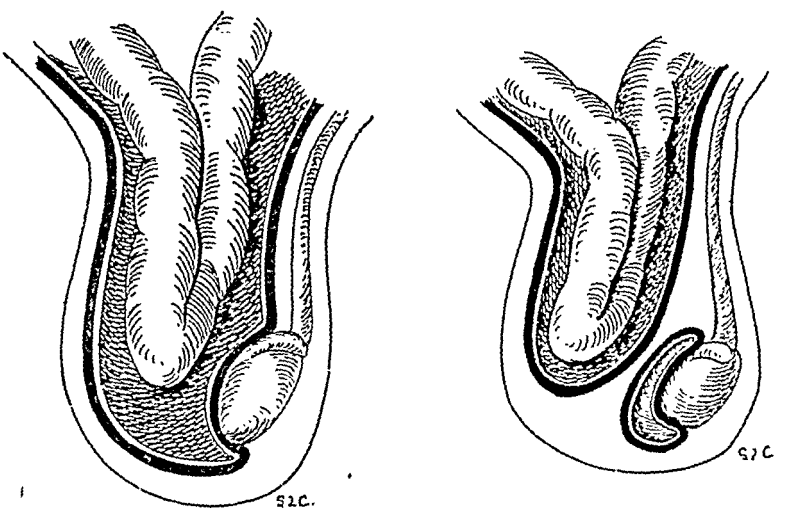

FIG. 6.—The left-hand illustration shows the relation of the testicle to the sac of a congenital scrotal hernia; the right-hand illustration shows the relation of the testicle to the sac of an acquired scrotal hernia. (Adapted from Gray.)

If the vaginal process has become obliterated below, but not above, there will be a channel extending from the abdominal cavity down to the site of obliteration. This channel is called the funicular process, and a projection of intestine into it is known as hernia into the funicular process. This variety differs from congenital hernia only in the extent to which it is possible for the gut to descend.

If the vaginal process is closed above, at or near the internal ring, but remains open below, two kinds of hernia may be formed. In some cases the septum between the abdominal cavity and the unobliterated vaginal process gives way slowly and allows the intestine to pass down behind the vaginal process. Such a hernia is known as an infantile hernia. Its sac is formed by the displaced septum together with the contiguous parietal peritoneum. The bowel has three peritoneal coverings, namely, the two walls of the vaginal process and the true sac of the hernia.

In other cases the septum gives way in such a manner that it and the contiguous parietal peritoneum are carried down into the unobliterated vaginal process. A hernia thus formed is termed an encysted infantile hernia. It has two peritoneal coverings, namely, the anterior wall of the vaginal process and its proper sac.[1]

Finally, a remnant of the vaginal process may persist above at the abdominal ostium, projecting slightly downward into the inguinal canal or appearing merely as a bulging at the internal abdominal ring. That such remnants predispose greatly to the formation of hernia is not to be doubted. In reality they constitute a preformed sac which becomes stretched and elongated by the pressure of the gut which impinges upon them from above. It is no doubt true that a considerable percentage of herniæ which are thought to be acquired originate in these remnants of the vaginal process, and therefore are originally due to a congenital defect. To maintain that all indirect herniæ are dependent upon such a defect, however, as has recently been done by some writers, is to ignore a very important structural condition normally present in the inguinal triangle which may rationally be considered to predispose to the escape of the intra-abdominal contents, namely, the arrangement of the transversalis fascia at the internal ring. This ring, although only a potential one, can be proved both embryologically and anatomically to constitute a weak spot in the parietes; and to deny the possibility and probability of this spot yielding to intra-abdominal pressure is, we think, unwarranted. Another point to be considered is the following: when a mere bulging of peritoneum is found at the internal ring in the adult subject, can it be positively determined whether this bulging represents a remnant of the vaginal process or the beginning of an acquired hernial sac? Certainly not. No one can prove it to be the one, no one can demonstrate it to be the other. The only way to decide the matter beyond question would be to cut a large number of children open in such a manner as to permit an examination for remnants of the vaginal process, sew them up again, and have them watched throughout life for the development of hernia. Until it shall have been determined in this manner that only those persons who possess remnants of the vaginal process develop hernia in adult life, we are unwilling to admit that all indirect inguinal herniæ are of congenital origin. We admit the probability of a considerable percentage being due to the presence of remnants of the vaginal pro-

[1] We believe that it would be better to abolish the old classification, according to which these herniæ are divided into congenital, infantile, etc., and call them all congenital. One is just as much dependent upon a defect of development as is another, and it certainly would be more accurate, and also probably less confusing to students, to speak of them as different forms of congenital hernia.

cess, but also believe that others develop irrespective of any defect in obliteration of this process.

Malposition of the testicle is a defect which is frequently associated with inguinal hernia. When the gland is retained high in the inguinal canal it has a tendency to dilate the internal ring and thus facilitate the escape of omentum and bowel. As the bowel passes downward its course may be obstructed by the testicle, so that it rises above the latter, with the result that the sac becomes curved. In cases in which the testicle has passed through the external ring, but has not descended into the lower part of the scrotum, the hernia pushes its way in front of the testicle, but usually does not descend below it. Non-descent of the testicle is also thought to be the chief cause of the rare pre-inguinal hernia, in which the sac escapes from the inguinal canal by pushing its way into the cellular tissue between the aponeurosis of the external oblique muscle and the skin. In this form the sac is divided into two or even three parts—one inguinal, one abdominal, and occasionally also a scrotal portion. The abdominal or superficial sac is the more important. It may be situated between the external abdominal ring and the anterior superior spine of the ilium, just in front of the ring, between the scrotum and the thigh, as in a case reported by Busch; or it may extend up toward the umbilicus, as it did in Broca's case.

Another variety most likely dependent upon a congenital defect is the inguino-interstitial, which develops in the inguinal canal, but works its way into the substance of the abdominal wall instead of emerging through the external ring, which is much smaller than usual or even absent. In the male such herniæ are associated with malposition of the testicle, and the narrowness of the ring was supposed by Tillaux and others to be due to the fact that the testicle had not passed through the external oblique muscle. Some cases, however, have been observed in the female, and, moreover, in others an accessory sac has been found in the scrotum, and the external ring has been observed to be wider than normal. Berger believes that the location of the sac in the substance of the abdominal wall is due to the presence of a congenital diverticulum in the vaginal process of peritoneum, which becomes enlarged by intra-abdominal pressure after the intestine engages in it. Such a hernia usually lies beneath the three lateral muscles of the abdomen, although some have been found between the internal and external oblique, extending high up into the abdominal wall.

Finally, there remains to be described the properitoneal hernia, for the pathogenesis of which a number of explanations have been offered. This variety of hernia also has two sacs, one deep and one superficial. The deep, or properitoneal sac is situated between the peritoneum and the transversalis fascia, and generally extends outward toward the anterior superior spine of the ilium. It may.

however, extend downward toward the pubes. The superficial sac occupies the inguinal canal and sometimes extends down into the scrotum, just as any other hernia which follows the cord may do. An angle of varying degree marks the divergence of one sac from the other and there is also a space, forming a vestibule, between them. Although some difference of opinion has existed as to whether properitoneal hernia is congenital or acquired, it has come to be pretty generally admitted that it owes its origin to a congenital defect. Thus, some have taught that the properitoneal sac is formed from a peritoneal diverticulum situated near the inguinal fossæ and that the superficial one is merely a spur carried out from the deep one by a process of dilatation as more and more of the intra-abdominal contents force themselves into it. Others maintain that both sacs are formed from the vaginal process, the deep from the iliac portion, which often presents a retro-inguinal fold well adapted for distension, and the superficial from the remainder of the process. The latter theory is the simpler and, moreover, is the one which those who have recently studied the subject most carefully are inclined to accept.

Perineal ectopy of the testicle may also be associated with hernia. This is infrequent, and is mentioned merely for the sake of completeness.

With regard to direct hernia it may be stated that this variety is never of congenital origin. The middle inguinal fossa is structurally a weak spot, through which the abdominal contents can be pushed by increased pressure from above. It has already been so well described under anatomy that further allusion to its structural peculiarities and relations are unnecessary.

Among conditions which increase the natural weakness of the abdominal wall may be mentioned diseases which impair the nutrition of the muscles, such as rickets, for instance; systemic maladies which produce great emaciation; abdominal tumors which impinge upon the anterior belly wall and distend it; pregnancy, which acts in the same way; accumulation of fat in the omentum and preperitoneal tissue, also tending to stretch the peritoneum; relaxation of the parietes incident to old age; and, finally, repeated efforts in lifting or dragging heavy weights.

It will be readily understood that some of these causes, such as pregnancy and accumulation of fat in the omentum, also increase intra-abdominal pressure. Other potent factors in augmenting pressure are cough, vomiting, and straining to empty the rectum or bladder. Bronchitis, emphysema, asthma, and whooping cough are not uncommonly the immediate exciting cause of hernia. In Berger's series of cases cough was considered an etiological factor more than eight hundred times. Elongation of the mesentery has also been considered a factor in the production of hernia. Although the mesentery is relatively longer in infancy than it is

in adult life, it is probably never so short as to prevent the intestines from coming out of the abdomen. This opinion is confirmed by the investigations of Mr. C. B. Lockwood, of London.

Age, sex, and heredity have also to be considered from the standpoint of etiology. In regard to age, it may be stated that hernia is most common in childhood and in middle adult life. Berger, who has studied the age incidence carefully, shows that by far the larger percentage of cases occur in children, that there is a constant decline until the thirtieth year, after which a steady increase takes place until the fifty-fifth year. It should not be forgotten, however, that hernia is relatively frequent in old persons on account of the relaxation of their abdominal wall and their susceptibility to bronchitis, asthma, etc. Were it not for the fact that they lead a shielded life and do not follow laborious occupations, they would be more liable to hernia than those in middle life. Berger's statistics also show that double hernia is rare in infancy and frequent in old age, reaching its maximum at sixty-five years.

All will admit that inguinal hernia is more common in males than it is in females. It is our belief, however, that it is more common in females than it has been thought to be, as the statistics of those hospitals and dispensaries in which female physicians are employed show a greater percentage of cases in women and girls than those institutions where only male physicians are in attendance. In over 800 operations for inguinal hernia which one of us has performed, not more than 50 were upon women.

The role of heredity is not thoroughly understood, but in our opinion it is of unmistakable etiological influence, as we know of many families in which several members have hernia.

Pathology. In studying the pathology of hernia the sac should receive consideration first. In shape it may be round, cylindrical, conical, or pyriform. In size it varies greatly. That portion just below the internal ring is called the neck, the remaining part the body. It differs in appearance according to the age of the hernia and the complications to which the hernia is subjected, being thin, smooth, and of the color of normal peritoneum in very recent cases; but thick, tough, corrugated, and white, yellow, or grayish in color, much resembling fibrous tissue, in those of long duration. In strangulation, it becomes dark red, purple, or black. In herniæ which have existed for any length of time it becomes blended with the other coverings of the bowel, frequently to such an extent that its separation is very difficult or even impossible. This is particularly true in regard to its relation with the transversalis fascia. In old herniæ there has usually been sufficient inflammation to blend the two layers inseparably together. One of us has had the opportunity of making postmortem dissection of a number of large scrotal herniæ, evidently of many years' duration, and has found that it is impossible to separate the coverings into more than two

layers after the herniated mass has been freed from the scrotum. One of these layers has invariably been traced to the aponeurosis of the external oblique muscle, the other has always disappeared within the internal hernial orifice. It is our opinion that the internal layer is composed of both peritoneum and transversalis fascia. In no case has it been possible to find any traces of cremaster muscle fibers. The external layer has always been adherent to the internal surface of the scrotum.

As the result of inflammation, which is bound to occur in the course of time, adhesions form which prevent the reduction of the sac with its contents. In the dissections just mentioned, the sac, together with other coverings, has been firmly attached to the pillars of the external ring, so that considerable cutting was necessary to free it.

Adhesions also not uncommonly form between the sac and its contents, a circumstance which still further renders reduction difficult or imperfect. Some of these inflammed areas may undergo calcareous change. Finally the inflammatory process may cause partial obliteration of the sac. It commences at the neck and may be confined to this part, but in some cases it extends downward and causes a variable degree of contraction. If obliteration at the neck be complete, a cyst may be formed in the lower part of the sac, owing to retention of the secretion which is produced. If a zone of obliteration occurs below the neck, the sac will be divided into two parts. These multiple sacs have been found both in congenital and acquired hernia. Longitudinal fibrous bands have also been observed. We have seen a few of these ourselves, and they have been mentioned by Okinczyc. No doubt others have also noticed them.

Hernia always produces changes in the abdominal parietes, the alterations depending upon the size of the hernia. The inguinal canal is always dilated, and the two abdominal rings are stretched and more or less approximated. The deep epigastric vessels may be displaced inward. Large herniæ cause pressure atrophy of the lower fibers of the abdominal muscles, and when they descend into the scrotum may likewise produce atrophy of the testicle. The extent to which the inguinal region may be weakened is well illustrated by the conditions which one of us recently observed in the case of a dissecting room subject who presented a large irreducible scrotal hernia which showed every evidence of having existed for years. The lateral muscles on the affected side in the lower part of the abdomen had become converted into a thin homogeneous layer, looking much like a piece of sheepskin; the aponeurosis of the external oblique had lost its firmness, presenting a number of small loose folds for a considerable distance above the anterior superior spine of the ilium, and the lower margin of the external ring had been pushed down below Poupart's ligament

almost to the saphenous opening. There was hardly any space between the superior margin of this greatly dilated external ring and the internal hernial orifice.

In reference to the contents of inguinal hernia, it may be stated that they are usually composed of omentum and small intestine. The large intestine, the stomach, the uterus and ovaries, the bladder, in fact any of the intra-abdominal contents but the pancreas may occasionally escape through the inguinal region. It is stated that a floating kidney has been found in the sac of an inguinal hernia.

The contents are subject to morbid changes. Thus the omentum is never normal except in very recent cases. Its adherence to the sac has already been mentioned. Its folds also become adherent to one another and sometimes portions of it become attached to the intestine. At the neck of the hernia the omentum is also often found converted into a fibrous band, this condition being the result of inflammation.

From a practical point of view the most important change which takes place in hernia is strangulation, by which is meant an occlusion of the lumen of the intestine together with arrest of its blood supply, produced by a constriction, which also renders the hernia irreducible if it was not already so before the said constriction occurred. As a result of strangulation the passage of both feces and gas is stopped and gangrene supervenes in the herniated bowel.

The most common site of strangulation in inguinal hernia we believe to be at the external abdominal ring, and not at the internal ring or within the sac, as has frequently been taught. Certainly this has been our experience, and we believe also that of most modern surgeons. In recent herniæ it may occur in the inguinal canal, between the two rings, being due probably to inflammatory changes in the transversalis fascia. In congenital hernia strangulation may be produced by strands or valves within the sac, or by the neck of the sac itself. In like manner it may result from the action of the inflammatory bands present in a partly obliterated sac, or be caused by the omentum, which, becoming adherent to the sac, forms a band that constricts the intestine. Rarely the bowel has perforated the omentum and become strangulated. Finally the possibility of torsion of the mesentery within the hernial sac must be borne in mind as a possible cause of strangulation.

The appearance of the strangulated intestine depends upon the length of time which the constriction has lasted. At first deep red, it gradually becomes darker and darker, until it may be almost or quite black. Its lustre disappears, patches of fibrinous deposit become visible, and here and there breaches in the continuity of the serous covering take place. These changes are the forerunners of gangrene, being shortly superseded by sloughs, which in turn may lead to perforation of the bowel. If the strangulation be relieved, the constriction of the intestine is plainly discernible.

The sac of a strangulated hernia is usually round in shape and almost always contains a considerable quantity of fluid. The sac, like the intestine, is much congested and may even share in the gangrenous process with which the latter becomes affected. We have often found the sac of a strangulated hernia to be of a dull slate color, with violaceous or dark brown patches scattered over its surface. The liquid in the sac is at first thin, clear, and of a light yellow color; but it soon becomes thick, turbid, and of a reddish or brown tinge, and contains flakes of fibrin. It may soon assume a fecal odor, caused by the presence of the colon bacillus, which migrates easily through the walls of the damaged bowel. Occasionally there is no fluid, the bowel being in direct contact with the internal surface of the sac. It is important to remember this fact and not to depend upon an outflow of fluid as a necessary sign that the bowel has been reached. Rarely it happens that only a segment of the bowel becomes constricted. To this condition the term lateral strangulation may be applied (Richter's hernia). Although the caliber of the bowel is diminished, it may nevertheless be possible for gas and fecal matter to work their way through. The bowel immediately below the constriction is usually found dilated, so that it may present the appearance of a diverticulum.

By an obstructed hernia is meant one in which the intestinal contents have accumulated to such an extent as to block the fecal current. The term incarcerated hernia has also been applied to this condition, but we believe it to be an inaccurate one, as it does not convey the idea of the condition which actually exists. If the term incarcerated is to be used at all, we believe it should be applied to those herniæ which are temporarily irreducible. We insist that this would be the correct use of the term. Obstructed hernia is probably much rarer than it was formerly supposed to be. Its existence has even been denied, but without sufficient reason, we are sure. We have seen the condition a number of times, and Nau has recently collected 23 authentic cases. It occurs almost always in herniæ of the large intestine, which have become irreducible as the result of inflammation. Intestine thus imprisoned loses its peristaltic power more or less, and this functional impairment, together with the mechanical conditions present, permit the occurrence of a gradual fecal obstruction.

A hernia may be attacked by tuberculosis or malignant disease. Cotte[2] has recently collected 136 cases of tuberculosis—the most thorough study of the subject with which we are familiar. The sac of the hernia alone, or both sac and contents, may be affected. Jonnesco states that the intestine may be diseased and the sac healthy, but his statement has not been generally accepted. In children the sac is usually the site of the tuberculosis; in adults

[2] Revue de Gynecologie, November and December, 1906.

both sac and contents are usually affected. Three forms of tuberculosis have been observed in the sac, namely, the miliary, the caseous, and the fibrous. Of these, the miliary is the most common. It is evidently the same lesion as that of miliary tuberculous peritonitis. There is generally considerable fluid in the hernial sac. Next in order of frequency is the caseous form, the fibrous being the rarest of all. The lesions of the intestines are no different from those found when the bowel becomes tuberculous in its normal habitat.

The neoplasms which have been found associated with hernia are carcinoma, sarcoma, and lipoma. They may affect the sac, the intestine, the omentum, or whatever organ is contained within the sac. The lipomas naturally develop in the omentum.

In another paper the diagnosis and treatment of inguinal hernia will be discussed.

THE ACTION OF THE SHORT ROTATORS ON THE NORMAL ABDUCTION OF THE ARM, WITH A CONSIDERATION OF THEIR ACTION IN SOME CASES OF SUBACROMIAL BURSITIS AND ALLIED CONDITIONS.

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of Boston, Mass.

Codman, of Boston, in a recent monograph on lesions of the shoulder-joint due, in his opinion, to trauma or to primary inflammation of the subacromial bursa, has done more to clear up the interesting subject of painful shoulders, and to explain certain definite restrictions of motion between the humerus and the scapula than has been accomplished by any other investigator, and his masterly interpretation of the mechanics of the shoulder-joint is so simple, clear, and conclusive (as far as he goes) as to establish almost beyond question his contention as to the primary action of the supraspinatus in abduction of the humerus preliminary to the action of the deltoid, which is thus permitted to come into action and exert its force in the proper direction to complete the arc of abduction. He has followed his theory through the operating room and into the dissecting room, and has conclusively established his contention by anatomical specimens showing the pathological changes following rupture of the supraspinatus, with the subsequent loss of the power of abduction in these cases. He has applied his researches into the normal mechanics of the scapulohumeral joint to the practical treatment of a class of shoulder lesions showing restriction of motion and tenderness below the acromion, which, as he says, constitute by far the largest class of patients applying to any

surgical clinic for the relief of trouble referable to the shoulder-joint. For this class of cases he has adopted Kuster's term of subacromial bursitis in place of the subdeltoid bursitis used by him in his first paper, although the latter term would seem to be by far the better, since more of the bursa is subdeltoid than subacromial, and since Piersol, in his recent anatomy, used the term subdeltoid in his description of the bursa. Whatever term is used, the credit must go to Codman for the most convincing description of the mechanics of this complicated joint. And in the light of his investigation the real action of the joint does not appear to be so complicated after all. A study of normal shoulder action is necessary for the proper understanding of his cases, and the few which have come under observation which we hope to demonstrate are departures from the regular types which he has described. If one considers anatomically the origin and insertion of the various muscles surrounding the shoulder-joint, and if one bears in mind the fact that the real capsule of the joint is formed by these surrounding muscles, and not by what is known as the capsule, with its ligamentous bands, which is a lax structure neither aiding nor opposing the normal motions of the joint, it would seem to be easy to assume, and equally easy to prove from a study of the anatomical structure, that the first few degrees of motion in abduction are due to the supraspinatus contraction, and is preliminary or preparatory, as it were, to the action of the deltoid.

Piersol says that the capsule of the shoulder-joint, among other uses, by means of its tense, firm under surface helps to prevent the arm from being raised beyond a certain point in abduction, but it is extremely doubtful if the capsule has any such action, although this one is by far the more probable of all the uses ascribed to it by the various anatomists, and it is probable that the capsule per se has very little retarding action on the movements of the joint itself. As Codman says, it is necessary to disregard our previous teaching and conception of the structure which has been called the capsule. The real capsule of the joint is made up of the muscles which surround it. Codman's contentions, which are, I think, accepted for the most part or will be so accepted by all who carefully examine the anatomical structure of the shoulder-joint, bearing in mind the laxity of the capsule, are, briefly, that the first few degrees of motion in abduction of the humerus are always due to the contraction of the supraspinatus acting on the short arm of the lever, the fulcrum of which is on the glenoid, the long arm of the lever extending downward through the arm (Fig. 1).

It will be readily seen that the fulcrum of such a lever must be a constantly changing point, and that the action of such a lever would be comparatively slight, and such seems to be the case. From its insertion high up on the greater tuberosity of the humerus the action of the supraspinatus must be limited in power, because the power in that case would be applied so near the fulcrum, while the weight

would be far removed (see Fig. 1: *A*, power, *C*, weight, of the lever *A*, *B*, *C*), and it is only sufficient to move the arm outward in abduction sufficiently to permit the line of pull of the deltoid (the mean line of the pull of all the fibers of the deltoid, assuming that these muscle fibers work together, which would be the line *H-E*, in Fig. 2) to fall superiorly to the fulcrum (*B*, Fig. 2) of the lever, in which case the deltoid could expend its force in abducting the arm still more, instead

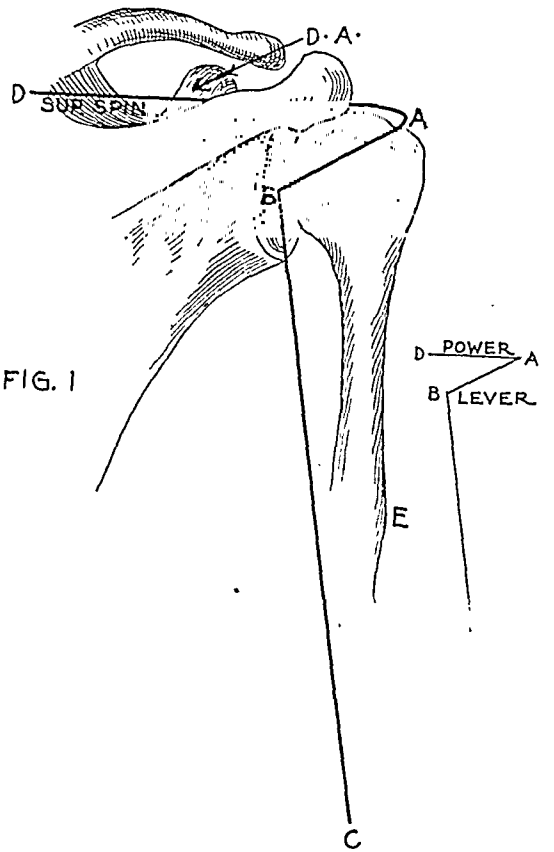

FIG. 1

FIG. 1.—*D A*, the line of pull of the supraspinatus; *A*, the power applied to the short arm of the lever; *A*, *B*, *C*, the lever; *B*, the fulcrum on the glenoid; *C*, the weight to be lifted. The contraction of the supraspinatus can swing the arm, obviously, in but one direction—abduction.

of uselessly forcing the humerus upward, as it would do acting in the line (*H*, *E*., Fig. 2) of its pull when inside (toward the body side) of the fulcrum on the glenoid, thus forcing the head of the bone against the lower surface of the acromion. It will be seen, therefore, from the origin and insertion of the deltoid (from the spine of the scapula, the acromion, and the outer third of the clavicle into the deltoid tubercle midway on the external surface of the shaft of the humerus) that its action alone, provided its fibers contracted as a single muscle,

would not be expended in the right direction to abduct the humerus, but rather would act uselessly in forcing the head of the bone against the under surface of the acromion, or if it worked too soon after

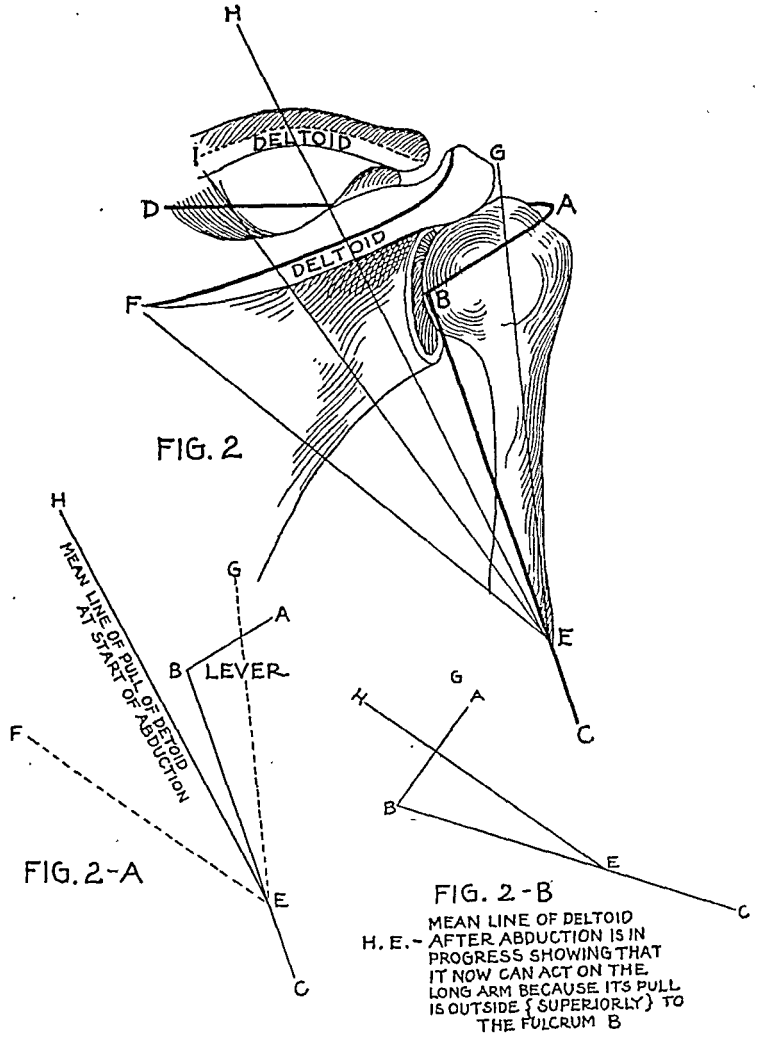

FIG. 2.—Showing the lines of pull of the fibers of the deltoid, providing these fibers acted together, H, E, being approximately the line of such pull, showing that in this case it could not act on the lever A, B, C, to abduct it. Assuming that the acromion portion of the deltoid acted as a separate muscle or as a separate contraction, then it could (consider the line G, E, and its effect acting at E on the long arm of the lever A, B, C) aid in starting the humerus upward in abduction, but should its fibers act simultaneously the line of pull would be inside the fulcrum, B (Fig. 2, A), and provided it was in contraction it would act in opposition to the line of pull of the supraspinatus, D, A. In order to act on the long arm of the lever the line H, E, must fall outside the point B (fulcrum), and this is possible only after the long arm of the lever (the humerus) has been partially abducted (Fig. 2, B).

primary action by the supraspinatus would force the greater tuberosity to take its fulcrum from the under surface of the acromion process (Fig. 2). If two lines of pull are at right angles to each

other, and equal force is applied in both directions, the body acted upon would always feel the force thus expended in a line midway between the two equally acting forces, and if one considers the origin and insertion of the deltoid it will be clearly demonstrated that if this muscle were acting alone it would not raise the arm in abduction (Fig. 2).

The normal mechanism of the shoulder-joint is, therefore, up to this point probably as follows: The supraspinatus contracting starts the motion of the humerus in abduction, but the supraspinatus from its insertion high up on the upper facet of the greater tuberosity of the humerus, is acting at a considerable disadvantage. It is the power applied to the short arm of a lever whose fulcrum is on the glenoid (Fig. 1, *B*, fulcrum, *D*, *A*, power). In other words, the lever is arranged, power, fulcrum, weight, the short arm of the lever being joined to the long arm at the fulcrum, at an angle which is constantly changing, and constantly becoming a more acute one as the arm rises in abduction, but which is at the beginning of motion usually a little over 45 degrees, and such a lever cannot, as said before, be one of great power. It is, however, sufficient to start the humerus outward, and begin the arc necessary to abduction, probably even up to 30 degrees, and during this time, unless the acromial portion of the deltoid acts as a separate and individual muscle from the rest of the deltoid, it can assist the action of the supraspinatus in no way (Fig. 2).

When the lever (humerus) has swung beyond a certain point the great deltoid contracting takes up the work, but it cannot do this until the supraspinatus has abducted the long arm of the lever (the humerus) sufficiently so that the deltoid contracting (the mean line of its pull when the muscles contract falling superiorly to the point of fulcrum on the glenoid) can act on the long arm of the lever, thus converting it into a more powerful one, where the power is applied as fulcrum, power, weight, and which is still aided by the power of the supraspinatus still acting on the short arm of the same lever. Should the deltoid contract before this point is reached, then the supraspinatus tendon is caught between the head of the bone and the acromion process, and either torn across or injured. As the arm reaches a certain degree of abduction the power of the supraspinatus, from its anatomical consideration, would seem to be slight, its work having been accomplished, but it would seem that its anatomical position is such as to render it of value as a tractor, possibly assisted by the lower tense part of the capsule (Piersol) to prevent slipping downward of the fulcrum (the articular surface of the head on the glenoid), thus preventing destroying the efficiency of the lever. If one looks at Fig. 3, he will see that this is the only part of the joint which is not surrounded by powerful tractors (the inferior portion), and motion here is prevented by the supraspinatus, tense superiorly, and possibly by the under surface of the capsule. If this should be

so, it would be the only value as far as aiding or retarding motion in the joint rendered by the so-called capsule. Another feature to be considered is the subacromial bursa which comes into play, as Codman clearly demonstrates, when the arm is abducted, and the tuberosity of the humerus is approaching the under surface of the acromion by the interposition of its well-lubricated surface between these two bony points (preventing the tuberosity from taking its fulcrum on the under surface of the acromion, and thus restricting further motion in abduction), permitting (the force of the deltoid continuing) the articular surface of the humerus to turn in but one direction, namely, towards the glenoid.

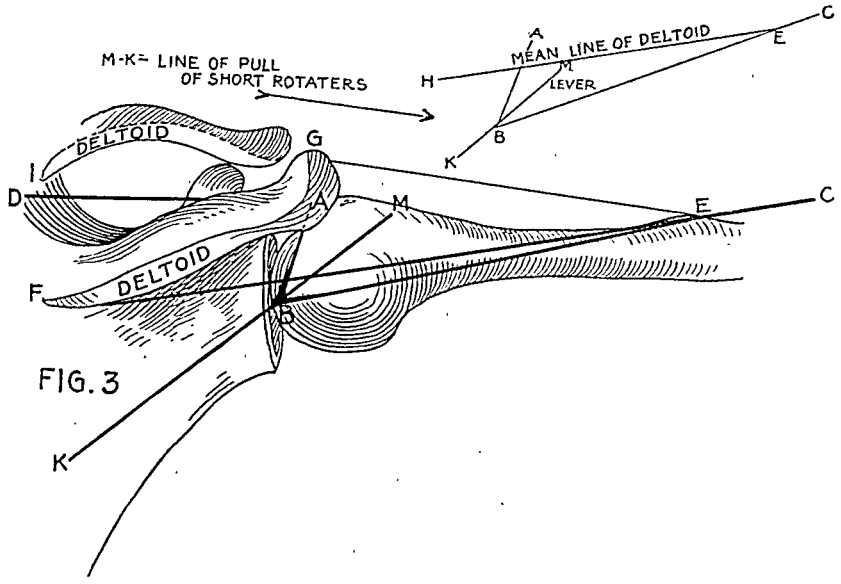

FIG. 3

FIG. 3.—This shows the lines of pull of the deltoid when the arm is partly abducted, G, E, F, E, and the line of the pull of the supraspinatus, D, A, acting on the short arm of the lever A, B, C, while the deltoid acts on the long arm of the same lever, its power being applied to E, a point between the weight C and the fulcrum B. Note also that both lines of pull, deltoid and supraspinatus, are above the fulcrum B, and unless there is another force applied, the fulcrum of the lever would be unstable and render worthless any amount of power applied. That force, K, M (the line of pull of the infraspinatus and teres minor behind and of the subscapularis in front) is supplied by these three short rotators, without which the head of the humerus would slip upward, answering the pull of the supraspinatus and deltoid. Note that the line of pull of the short rotators passes through the fulcrum, therefore it acts merely as a tractor and exerts no force either on the long or short arm of the lever, consequently offering no impediment to the abduction, while at the same time rendering such action possible by keeping the fulcrum firmly in its place.

Codman's contention is that the subacromial bursa accomplishes this purpose, and he goes no further. If we consider now the origin and insertion of these two muscles together, both acting and the humerus partly raised in abduction, we come to the consideration of the third part of the mechanics of the shoulder-joint in abduction, a very important part, indeed, and one which has not been considered

(so far as I know), but which I believe absolutely essential to the normal action of the joint, and which action is especially to be remembered in the class of cases classified by Codman, and also in those cases the consideration of which I shall take up, and which are closely allied to the condition known now as subacromial bursitis. As the arm continues to abduct, the line of pull of the supraspinatus and the line of the deltoid pull are coming nearer and nearer into line (Fig. 3, *D*, *A*, and *F*, *E*), and they are both upward and inward, and both forces are superior now to the point of the fulcrum (Fig. 3, *B*). Unless there were another force to offset this upward and inward pull of these two muscles, the tendency would still be to force the tuberosity against the under surface of the acromion process, and it is inconceivable that nature should leave such a task to the slippery surface of any interposed bursa. While, undoubtedly, this interposition of the bursa is necessary, and acts to direct the articular surface of the humerus against the glenoid, and also permits the tuberosity to pass smoothly under the acromion, still, without the presence of another force acting at an entirely different angle than either the supraspinatus or the deltoid, the articular surface, while it might and probably would be directed to the proper fulcrum, would not be held firmly enough to permit of that efficiency which is present in the normal joint, and it would permit riding up of the tuberosity against the acromion with unnecessary force.

Consider the lines of pull of these two muscles inward and upward, and we can see that the tendency would still be to pull the tuberosity upward either with or without the bursa (Fig. 3). Another force applied here is an absolute necessity. Such a force is supplied when we consider the origin and insertion of the short rotators.

Following the anatomical law that the strength of opposing muscles, or the strength of the sum of opposing muscles, is equal or nearly equal, then the subscapularis and the infraspinatus, plus the teres minor, must be equal, the one an inward rotator, the others outward rotators.

But consider the insertion of these muscles so near the head of the bone, and it becomes more probable that the simultaneous contraction of these three muscles, following the primary pull of the supraspinatus and simultaneous with the contraction of the deltoid, is necessary to pull the head of the humerus into the glenoid, and fix it firmly there forming the fulcrum, and that without this additional pull the action of the deltoid would still be to force the tuberosity to take its fulcrum from the under surface of the acromian, and that this action is equally important with their well-recognized action as rotators of the arm. These three short rotators or tractors, as they should be called, form a practical sling around the anatomical neck of the humerus, and their lines of pull, when acting together, would not only enable the articular surface to find its proper fulcrum, but would also counteract the tendency of the combined action of the

supraspinatus and the deltoid to pull upward the tuberosity (Fig. 3). Consider these muscles in their relation to the arms of the lever, and we see (Fig. 3) that the line of pull of these short rotators passes practically through the fulcrum of the lever, and, therefore, while exerting power sufficient to keep the articular surface constantly and firmly against the glenoid, and the tuberosity away from the acromion, thus permitting abduction of the humerus, the line of pull, passing as it does practically through the fulcrum, it could not in any way act on either arm of the lever to hinder elevation or depression of the humerus. For a demonstration of this contraction of the subscapularis, infraspinatus, and teres minor, it is only necessary to stand behind a model who is abducting his arm, and note with the hand the tenseness of these muscles, especially after the humerus has swung past the horizontal line, in contrast with the flabby relaxed belly and tendon of the pectoralis major in the same position. Beyond a certain point, however, the infraspinatus and teres minor behind and the subscapularis in front, which have acted together up to this point solely as tractors to hold in place the articular surface of the humerus, may possibly take on an added action, although it is probably to a very limited extent, inasmuch as abduction of the humerus is by this time a nearly completed act, namely, that of abductors also, as aids of the deltoid.

The action of the supraspinatus is practically little by this time, and the short arm of the lever has ceased to be of much advantage. Its force is probably expended more as a tractor to help hold firmly the articular surface of the humerus from slipping on its fulcrum, and it is in this possibly aided by the tense, firm inferior surface of the capsule, as suggested by Piersol. A new lever, therefore, takes the place of the old (Fig. 4). Considering the articular surface of the humerus as the segment of a circle, turning constantly as the arm abducts and forming a constantly changing fulcrum or point on the glenoid, the moment that the lines of pull of the short rotators have swung upward through the fulcrum of the old lever they cease to act simply as tractors, and a new lever, or a new short arm, is formed instantly. Or, whenever the line of pull of these short rotators transects the segment of the circle above the fulcrum they become the power on the short arm of a new lever, the long arm of which remains the same (Fig. 4, *M*, *B*, *C*), and their action would be to raise the arm to a still higher level, assisting the deltoid.

The reverse of this is also true to a limited extent. Thus, the short rotators act in abduction of the humerus when the arm has passed a certain level, and act to pull it downward, adduct, after it has fallen below another fixed level on its downward course, depending on the relation of the line of pull to the fulcrum of the lever at *B*.

To recapitulate, it would seem that from a theoretical and practical consideration of the shoulder-joint, always taking into consideration the origin and insertion of its various muscles, and considering

them as the power applied to the lever (the humerus), whose fulcrum in the normal shoulder would be a constantly changing point, it would seem that the necessary movements would be as follows: (1) Supraspinatus contraction would abduct the arm and tend to tip the articular surface of the humerus, so as to bring it against the glenoid, but its action and its strength are slight (30 degrees). The powerful deltoid, contracting, forces the tuberosity against the under surface of the acromion, where interposes the well-lubricated subacromial bursa, which, the force of the pull continuing, permits the articular surface to turn in but one direction, namely, toward the glenoid.

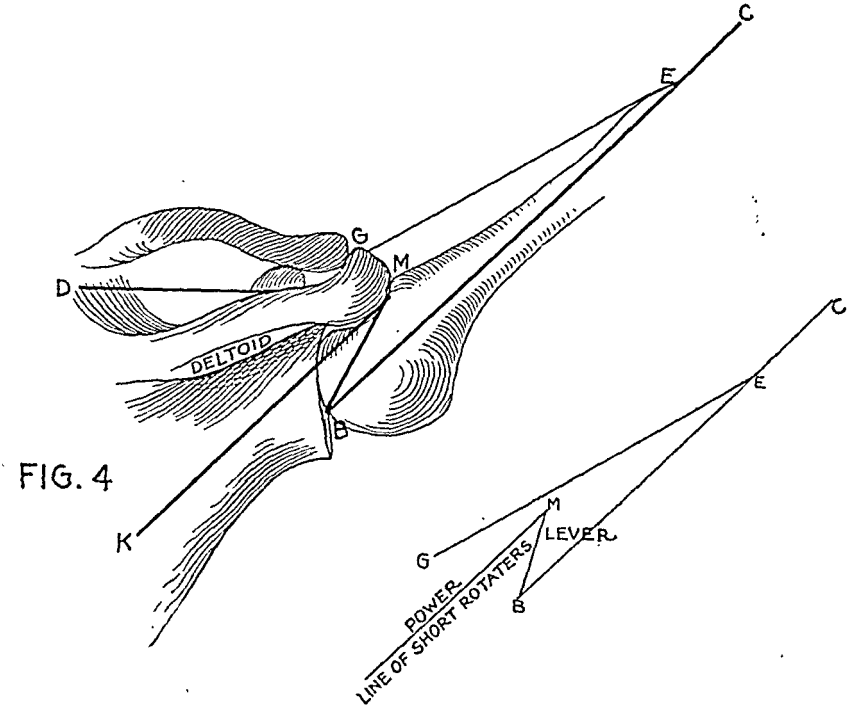

FIG. 4.—*G, E*, the power applied by the deltoid at *E* to the lever *M, B, C; M*, the point where the power is applied to the short arm of the new lever by the short rotators; *B*, the fulcrum on the glenoid; *K, M*, the line of pull of the short rotators. Note that the line of pull of the short rotators (subscapular, infraspinatus, and teres minor) is in this position of the arm superior to the point of fulcrum *B*, therefore it may act slightly as an additional abductor.

To complete this action and in order to form a theoretically perfect mechanical entity, we must invoke another force or pull, fixing the head of the bone in the glenoid, yet having no restriction on the abduction of the arm. Such a force we have seen that we have in the combined and simultaneous action of the subscapularis on the one side, and the infraspinatus and teres minor on the other. In other words, a narrow, firm sling pressing the head of the bone into

the glenoid, yet not preventing abduction, but even acting above a certain point as an aid, and varying in forward and backward swinging exactly as the muscles of one side over or under acted in consideration with the opposing muscles, always, however, exerting sufficient pull to keep the articular surface firmly against its fulcrum on the glenoid cavity of the scapula.

To come now to a consideration of the subject of shoulder lesions involving the bursa, or of lesions presenting symptoms which closely ally them to lesions of the subacromial bursa. Codman has investigated this subject thoroughly, and has written a monograph on the subject of subacromial bursitis which will become a classic. He has divided his cases into three classifications:

Type I, the acute or spasmodic form with localized tenderness just below the acromion process of the scapula, over the bursa, and to the outside of the bicipital groove. This tender point disappears under the acromion on abduction because no adhesions are present in the bursa to prevent, and this sign is considered pathognomonic of this type of bursal lesions by him (Dawbarn's sign). In abducting or in external rotation, after a certain point is reached the scapula is locked by spasm, and moves with the humerus. In mild cases, with but little spasm, the patient thinks he cannot abduct his arm, but will allow passive motion, and is usually perfectly able to abduct once he has overcome his fear of pain. Codman thinks this is explained by the unwillingness of the supraspinatus to start the pull on its sensitive tendon.

In *Type II* (the subacute or adherent type) actual adhesions exist in the bursa, and there is an actual mechanical limitation to abduction and external rotation. Localized tenderness may or may not be present, according to the degree of existing inflammation. Dawbarn's sign is absent—the tuberosity cannot pass under the acromion because of adhesions. Abduction and external rotation of the humerus on the scapula are limited; 10 degrees of free motion in abduction exist because the bursa is not brought into play before this point is reached. Beyond this arc of 10 degrees the scapula accompanies the humerus in all its motions, active or passive. Pain is in the same distribution as Type I, and in severe cases it may resemble a brachial neuritis.

Type III. The chronic type; the essential characteristics are painful motion, but the full arc exists. The trouble is due in this class to slight irregularities of contour of the base of the bursa usually external to the bicipital groove. Motion, instead of being smooth, is jerky and interrupted in its arc. Localized tenderness may or may not be present. If present, then Dawbarn's sign is present. Abduction and external rotation are but slightly interferred with, but at some point in abduction acute tenderness is experienced, which disappears as soon as the tuberosity is under the acromion. The scapula does not accompany the humerus. There may or

may not be pain, and if present it is often felt at the insertion of the deltoid.

While forced to agree with all which Codman says in his paper, as far as he goes, and also that nearly all of the shoulder cases will fall into one or the other of his classification, it has been my fortune recently to come in contact with two cases, out of a total of seven, which it seems to me impossible to class with any one of the three types which he has given, and it has seemed that there was possibly a class of cases, limited in number perhaps, and usually or always the result of trauma, the symptoms of which are practically those of subacromial bursitis, and which may be, and perhaps always are, accompanied by some inflammatory changes of the bursa, but which show distinctive symptoms sufficient to call attention to the involvement of the short rotators, the action of which Codman does not take up in his article.

These two cases, one following a dislocation unreduced for twelve hours, and the other a fall with the arm in extension, presented the same symptoms: No tender point over the bursa beneath the acromion; the greater tuberosity disappeared under the acromion; a normal active arc without pain up to 23 to 25 degrees, and then restriction, but not on account of pain; passive motion, practically throughout the entire arc; external rotation lost, internal rotation preserved; no paralysis of the deltoid.

From an examination of these cases, I am forced to the conclusion that the greater number will readily fall into Codman's classification, and that they will also show a subacromial bursitis with or without adhesions; that at autopsy evidence of tearing or fraying of the tendon of the supraspinatus will be more often observed than evidence of injury to the short rotators, but it is my contention that there is a limited number of cases which, with or without a well-defined bursitis, present certain varieties of symptoms which are not due entirely to inflammation or adhesion of the bursa, but which are due to either a tearing or more frequently an inflammatory involvement of these short rotators, more particularly the infraspinatus and the teres minor. There is no tender point over the bursa. In the acute cases of Codman there is a tender point. The greater tuberosity disappears under the acromion without pain. In his cases of acute type the tuberosity disappears under the acromion, but with pain, and in the adherent type it does not disappear at all. His cases show 10 degrees of abduction before pain. The cases under observation show 23 to 25 degrees of active abduction, and then motion was not stopped on account of pain; it was inability to lift the arm, the deltoid visibly acting. Inward rotation was preserved, and this would apply equally well to bursitis alone, the preservation being due to less involvement of the subscapularis, because it comes to a less degree into contact with the bursa. External rotation was lost, and Codman, contradicting Kuster, says that it is also true of subacromial bursitis,

probably accounted for by the proximity of the posterior short rotators to the floor of the tender bursa.

In the two cases cited there is, however, no tender bursa, at least not on pressure, passive motion is practically preserved, which would not be the case in adherent bursitis, and would be painful in acute bursitis, while in his chronic type external rotation is preserved. The full arc of passive motion, the absence of tender points over the bursa, the preservation of internal rotation, the loss of external rotation, 23 to 25 degress of painless, active abduction, and no deltoid paralysis, together with tenderness over the bellies of the infraspinatus and teres minor, and especially marked and referred outward to their insertion on using faradization, would seem sufficient to warrant us in recognizing a lesion of the shoulder-joint usually following trauma, which may be accompanied by a low grade of bursal inflammation, and may always be so accompanied, but whose chief pathological change is either a complete or incomplete rupture, or, more frequently, the involvement in an inflammatory lesion of the tendons of the infraspinatus and teres minor, and less frequently of the susbcapularis.

There is also a class of fractures involving the greater tuberosity of the humerus, not so rare as has been assumed, which invariably lead to a set of symptoms resembling bursitis. Some of these cases undoubtedly develop a bursitis, and to that condition their symptoms should direct attention, but some of them present symptoms identical with those described in the two cases reported above, and it has seemed to me that involvement of the short rotators, which can be proved by the *x*-rays, is more of a factor in these particular cases than an inflammation of the bursa. In the two cases which have presented themselves to me, in which the accompanying *x*-ray prints show the nature of the lesion, there has been some question both among the clinicians and the *x*-ray men as to the exact anatomical injury, but it will be plain to anyone, without entering into a discussion of that point, that the greater tuberosity has been, at least, torn off.

In Fig. 5 it shows as an absolute loss of substance, and for the purposes of this paper it is sufficient to deal with it simply as a lesion of the tuberosity. The loss of motion to have been expected here was absolute inability to abduct and loss of external rotation with the preservation of internal rotation. This was exactly what this patient showed. He could move his arm in the frontal plane because his biceps was not involved, but he could not start his arm from the side in abduction because his supraspinatus was torn loose, and external rotation was lost because the same thing had happened to the infraspinatus and teres minor. The deltoid was not involved, and could be seen to contract vigorously both voluntarily and by faradization, but the arm was not abducted, and could not be held when passively abducted. Internal rotation was preserved because the position of the subscapularis was not involved in the injury and its belly and

insertion were both intact. In this class of cases when passive motion is free and full, and the only restriction is in active motion, should there exist the 10 degrees of normal supraspinatus abduction

FIG. 5.—Fracture of the great tuberosity of the humerus, involving the insertions of the supraspinatus, infraspinatus, and teres minor, with displacement of the fragment. There was entire loss of abduction; external rotation was lost permanently, but the power to abduct was recovered; internal rotation was preserved; forward and backward motions were possible with slight pain. (*X*-ray by A. W. George, M.D.)

mentioned by Codman, then it is quite clear that that tendon has not been torn away, and if the motion is painless, it is equally certain that it has escaped injury.

If the humerus is abducted painlessly to 23 or 25 degrees and the deltoid contracts and still active abduction is impossible beyond that point, with the full arc on passive motion, and the disappearance of the tuberosity under the acromion, then the cause of the impairment of motion is, I am convinced, to be looked for in a lesion of the short rotators. If a painful point exists (Dawbarn's sign) it is possible that a bursitis is the cause of the loss of motion, but I am con-

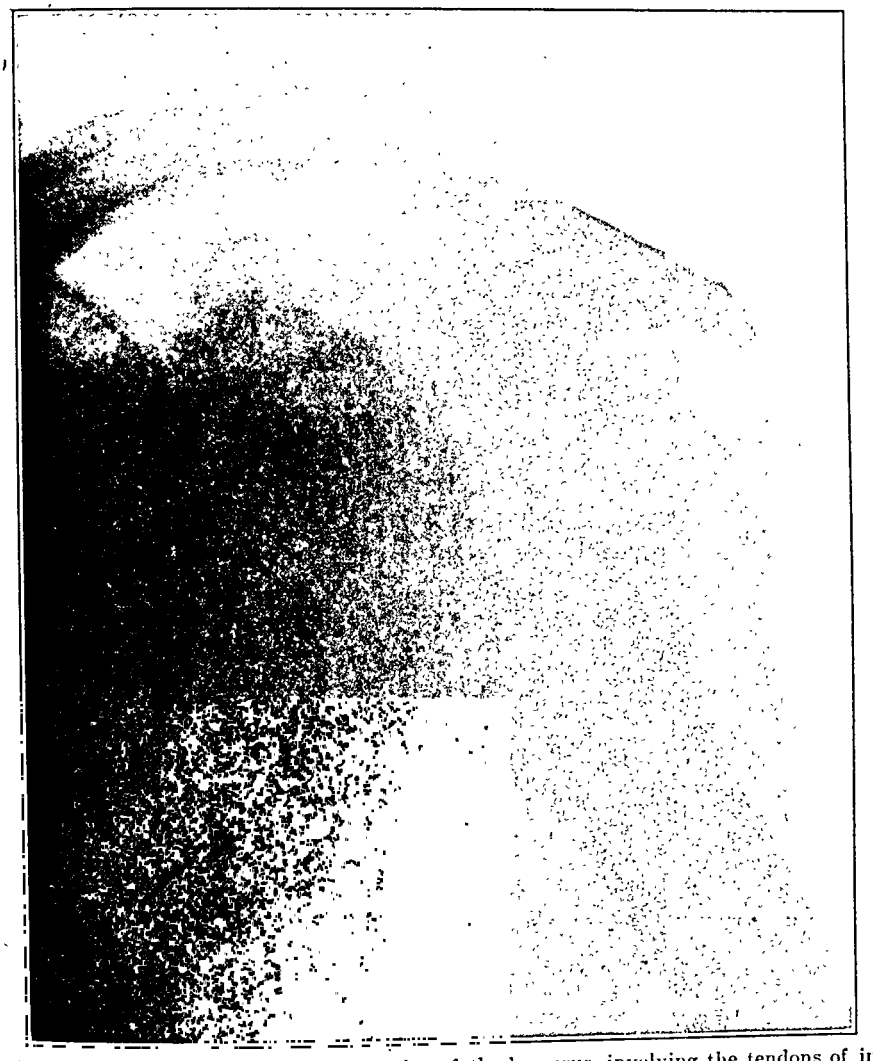

FIG. 6.—Fracture of the great tuberosity of the humerus, involving the tendons of insertion of the supraspinatus, infraspinatus, and teres minor; radiogram two and one-half months after the injury. Six months later external rotation was still lost. Internal rotation was preserved at all times. Some restriction in abduction still persists when the arm swings beyond the horizontal position. There is no pain; note callus. (*X*-ray by A. W. George, M.D.)

vinced that there is a certain number of cases with the symptoms mentioned in which the lesion is one involving the short rotators, more frequently the infraspinatus and teres minor, and that this is the principal lesion, whether or not associated with a bursitis. Among the

cases which Codman cites is one in which passive motion was free and the tuberosity disappeared under the acromion, but with tenderness. The deltoid, to quote his own words, was as big as a ham, and yet when abducted passively he could hold his arm only by tremendous effort, and then only for a few seconds. The weight of a finger would send it downward. It seems as though this might be equally characteristic of his first or third type, or of the class of cases to which I have called attention, and that the mechanism involved here is that either from injury or from their proximity to the base of the tender bursa the short rotators refused to do their work, therefore not holding the fulcrum firmly in place and permitting it to slip on the

FIG. 7.—Fracture of the great tuberosity of the humerus, involving the insertions of the supraspinatus, infraspinatus, and teres minor. The patient could start the arm in abduction only a few degrees, however, and with pain; motion forward and backward was preserved but restricted; external rotation was lost (involvement of the infraspinatus and teres minor); internal rotation was preserved (subscapularis intact). All motions were finally recovered. (*X*-ray by A. W. George, M.D.)

glenoid, in which case, a very large deltoid would be of as little value as one of microscopic size. A lever to be of any value must have a fixed fulcrum, and it must be, for the time being, at least, a firm and solid one. Disturb it in the slightest degree or render it unstable, even for the infinitesimal part of a second, and unless one instantly substitutes another point for the one disturbed, the greater the force at the long arm of the lever the quicker and more surely will that lever fail.

SPASTIC PARAPLEGIA DATING FROM CHILDHOOD (LITTLE'S DISEASE?), WITH LITTLE OR NO DEMONSTRABLE LESION IN THE PYRAMIDAL TRACTS.

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It is not the object of this paper to discuss the classification of cases of cerebral spastic palsies, or the relation which Little's disease bears to these cases; but rather to call attention to the remarkable and interesting fact, already discussed by Spiller[1] and others, that spastic paraplegia may occur with little or no demonstrable degeneration in the pyramidal tracts of the cord, and to add a case in point.

While a few cases have been reported bearing on this subject, the literature is not very extensive, and the last word has not yet been said. Although in some cases degeneration of the pyramidal tracts has been described, this has been caused either by a spinal lesion (Dejerine,[2] Spiller), a meningo-encephalitis (Sachs[3]), a sclerosis of the motor cortical areas (McMutt[4]), or a venous effusion into the meninges, causing destruction of the cortex (Muratow[5]). The cases of porencephalon and atrophy of the motor and other convolutions, without degeneration in the pyramidal tracts (Railton,[6] Ross,[7] Phillip and Cestan[8]), present a different problem; and yet more difficult of explanation are those cases in which there is little or no demonstrable lesion, either in the cortex or the motor tracts of the spinal cord, as in the cases of Spiller, Rolly,[9] Dejerine, Haushalter and Collins,[10] and in my own case to be reported herewith.

In some of the cases no true degeneration of the pyramidal tracts is found, and in these have been described: a fineness of the fibers of the pyramidal tracts (Spiller, Biswanger[11]), a scarcity of fibers (Ganghofner,[12] Gerlich[13]), incomplete myelization without diminution in the number or size of the fibers (Haushalter and Collins), agenesia or imperfect development of the pyramidal tracts (Dejerine, Spiller, Anglade, Jacquin,[14] Ganghofner, Cestan,[15] Steinlechner-Gretschisch-

[1] University of Pennsylvania Medical Bulletin, January, 1905; Jour. Nerv. and Ment. Dis., 1898, 81.
[2] Revue neurol., 1903, xi, 601. Comp.-rend de la soc. de biol., 1897, p. 261.
[3] New York Med. Jour., 1891, v, 1.
[4] Amer. Jour. Med. Sci., 1885, 59.
[5] Deut. Ztschr. f. Nerv., 1896-97, x, 272.
[6] Brit. Med. Jour., 1892, i, 441.
[7] Brain, 1882, 473.
[8] Comp.-rend. de la soc. de biol., 1897, p. 1080.
[9] Deut. Ztschr. f. Nerv., 1901, xx, 152.
[10] Comp.-rend. de la soc. de biol., 1905, lix, 223.
[11] Virchow's Archiv, 1882, 427.
[12] Ztschr. f. Heilk., 1896, xvii, 203.
[13] Arch. f. Psych., 1891-92, xxiii, 201.
[14] L'encephale, 1909, 252.
[15] Prog. Med., 1899, x, 102.

nikoff,[16] and Oddo[17]) or atrophy of the pyramidal tracts (Ganghofner, Steinlechner-Gretschischnikoff, Oppenheim[18]); while an effort has been made to connect some of these conditions with degeneration of the cortical ganglion cells, or diminished number, or absence of these (Biswanger, Rolly, Otto,[19] Köster,[20] Sachs).

This brief and hasty review of the pathology of the cases of cerebral spastic palsies dating from infancy or birth, does not explain satisfactorily at once the cause of the spasticity, and the paralysis in all the cases. Marie's theory that the contractures are due to absence of the pyramidal tracts, which normally exercise an inhibitory influence on the cells in the anterior horns, is not applicable in those cases in which little or no change can be demonstrated in the pyramidal tracts. Mya and Levi[21] believed also that the contractures in Little's disease were entirely of spinal origin, resulting from the predominance of the action of the cells of the anterior horns in agenesia of the pyramidal tracts. They believed that in the newborn the radicular cells of the spinal cord have an autonomous function, independent of the cortical cells. The physiological hypertonicity at this age is accentuated when the motor centres of the cord are not properly connected with the higher centres. Van Gehuchten,[22] however opposed this view, claiming that the question of age is of little importance, as the same spasticity is found in adults in affections of the pyramidal tracts. He believed that the contractures are an expression of an exaggeration of normal muscular tone, due to an interruption of the corticospinal fibers, with persistence of the corticopontocerebellospinal fibers, maintaining the cells of the cord under the influence of the motor cells of the cortex.

It is not difficult to understand that sparseness of fibers, or fineness of fibers, may have the effect of lowering conductibility, and in this way interfere with the inhibitory control normally exercised by the cortex over the spinal centres, the persistent action of the cells of the anterior horns thus giving rise to spastic conditions of the extremities.

One word as to the cause of the agenesia of the pyramidal tracts, the presence of poorly developed, thin fibers, and the sparseness of fibers in the pyramidal tracts, described in some of the cases. That these conditions result from a lack of the usual number of cortical ganglion cells is plausible, although extremely difficult to demonstrate satisfactorily. The view held by Van Gehuchten is also acceptable in this connection: that spasticity is due to the arrest of growth of the fibers in the pyramidal tracts, he having found, in fœtuses of seven months, that the axis cylinders are absent in the cord, but present above the anterior pyramids.

[16] Arch. f. Psych., 1886, xvii, 649.
[17] Arch. d. méd. d'enf., Paris, 1899, ii.
[18] Arch. f. Psych., 1891-92, xxiii, p. 53.
[19] Cited by Oddo.
[19] Neurol. Centralbl., 1895, p. 130.
[20] Neurol. Centralbl., 1887, No. 10.
[22] Revue neurol., 1897, 558.

How can the spasticity be explained when the pyramidal tracts are not degenerated? The presence of fibers in the pyramidal tracts presupposes the presence of intact cells in the cortex, or at least in the cerebrum. The character of the fibers and cells, then, must have some bearing upon the development of this symptom. When fibers of small caliber are called upon to conduct vigorous motor impulses, do we not meet with the same condition as when large currents of electricity are forced through wires of small caliber, that is, increased resistance, and imperfect conduction? The increased resistance offered by the small fibers to the motor impulses may prevent the impulses from reaching the cells in the spinal cord in toto, and thereby there is a cutting off, partial at least, of the influence of the higher inhibitory cortical centres. While, of course, this cannot be demonstrated, it seems a possible explanation of the spasticity in some cases.

The following case is interesting in this connection on account of the absence of gross and microscopic changes in the nervous system to account for the symptoms presented during life:

J. C., aged sixty-four years, was admitted to the Philadelphia Home for Incurables September 28, 1899. His father was killed in an accident, and his mother died as a result of a "broken blood-vessel." Three brothers are dead, two as the result of "kidney trouble;" the cause of death of the third is unknown. When a child the patient had measles, chickenpox, and "spasms." No history could be obtained as to the conditions at birth or during early infancy.

He stated that the present disease began at about five years of age, although he said, on a subsequent occasion, that the spasms in infancy had left him in his present condition. Physical examination showed a spastic condition of both legs, which remained stationary from the time of his admission (September 28, 1899) until his death (May 9, 1906). His gait was that of spastic diplegia, and he assumed a stooping attitude when walking. He assumed a perfectly erect position only with considerable difficulty, and could not maintain it for any length of time. The knee-jerks were much increased and equal. The Babinski phenomenon was marked on both sides, as was also the plantar reflex. There was no clonus present. His grasp in the right hand was good, but was slightly weaker in the left hand. He used his hands readily, and there seemed to be no loss of power or spasticity on either side. He spoke with difficulty, but this was due to stuttering, and there was no evidence of spastic speech. Mentally he was considerably below the average intelligence.

The autopsy was performed a few hours after death; the brain and spinal cord were removed and placed in formalin.

Macroscopically the brain showed an extensive effusion of blood in the pia arachnoid, extending over a large part of both hemi-

spheres, and also at the base. There was no evidence of fracture of the skull or local trauma. Otherwise the macroscopic examination was negative.

Sections of the brain and spinal cord revealed absolutely nothing abnormal macroscopically. Portions of both paracentral lobules and the left frontal region were studied microscopically, as well as the pons and medulla and various portions of the spinal cord. The pia of the cortex showed a sparse cellular infiltration composed of connective tissue cells. The bloodvessels were somewhat thickened, and the cellular elements in the walls somewhat increased.

The cells of the cortex, stained by thionin, revealed no abnormality, although no Betz cells were found. The pons, medulla, and spinal cord, stained by the Weigert method, showed no evidence of degeneration. Sections of the spinal cord stained with hematoxylin and acid fuchsin, showed that the crossed pyramidal tracts on both sides took the stain slightly more intensely than the remaining white matter. Study of the fibers in the crossed pyramidal tracts by this stain demonstrated the presence of a number of small fibers closely packed together, and a sparseness of large well-formed fibers. There was a slight increase in the connective tissue, but at no time did this reach the degree which is seen in spinal cords in which the pyramidal tracts show secondary degeneration. The smallness of the fibers of the crossed pyramidal tracts contrasted clearly with the much larger fibers in the adjoining direct cerebellar tracts. The cross section of the spinal cord itself was normal in size. There were also present in the crossed pyramidal tracts, as well as in the posterior columns at all the levels studied, numerous corpora amylaceæ. The fineness of the nerve fibers was more apparent in the cervical and thoracic regions than in the lumbar region. By the thionin stain the ganglion cells of the anterior horns, at all the levels studied, showed a marked change. While there were present many normal cells, a number of cells were seen which were swollen, some showed marked atrophy of the pigment with excentric nucleus, while most of the cells showed increase in the yellow pigment. At some levels fresh capillary hemorrhages were present in the posterior horns, probably agonal in origin.

SUMMARY. A man, aged seventy-one years, presented spastic paraplegia dating from early childhood. A study of the brain and spinal cord revealed nothing of importance, excepting a fineness of the fibers in the crossed pyramidal tracts.

In my opinion this case antedated the age of five years (as the patient stated), as his intelligence was below normal, and it could not be expected that at the age of sixty-four (when he was admitted) an accurate history of his early infancy could be obtained. The result of the anatomical study makes it possible that this case was one of Little's disease, although, of course, without definite history of the onset, this claim cannot be definitely made. It is certainly

extraordinary that an individual could live to the age of seventy-one years, suffering for certainly the greater part of his life from spastic paraplegia, without its being possible to demonstrate more pathological change in the nervous system than the slight, and perhaps indefinite one which I have described. It seemed to confirm the theory, advanced earlier in this paper, that the fine caliber of the nerve cells offered resistance to the transmission of motor impulses, which, for their perfect conduction, require well-formed and large nerve fibers.

The three cases reported by Spiller are apparently similar to mine. In two of his cases he found practically nothing abnormal in the nervous system except a fineness of the nerve fibers of the crossed pyramidal tracts, and in the third case there was no positive microscopic lesion whatever. He believed there was an agenesia of the pyramidal tracts in the spinal cord in the first two cases cited, while in the third one the explanation offered was that the nerve fibers had not grown downward as far as the lumbar region in normal numbers, although he confessed that this was difficult to prove.

REVIEWS.

THE PRINCIPLES AND PRACTICE OF MEDICINE. By WILLIAM OSLER, M.D., F.R.S., Regius Professor of Medicine in Oxford University, England. Seventh edition; pp. 1143. New York and London: D. Appleton & Co., 1909.

IT is a pleasure to record the publication of a new edition of Osler's *Practice of Medicine*, a book which, in many respects, ever since its original issuance, has not acknowledged a peer. The volume has been notably improved, particularly, as the author points out, in the section relating to the infectious diseases. Herein are incorporated the advances in the etiology of syphilis, the work of the New York Pneumonia Commission, the triumph of the British army and navy surgeons in stamping out Malta fever, the work of Gorgas and his colleagues at Panama in connection with yellow fever, the studies of Strong and his associates in the Philippine Islands in connection with dysentery, recent work in trypanosomiasis, psorosomiasis, tropical splenomegaly, cerebrospinal fever, tuberculosis, epidemic anterior poliomyelitis, Rocky Mountain fever, milk sickness, serum disease, carriers in acute infections, parasitic infections, etc. New sections have been incorporated on diverticulitis, parotitis, pancreatic and adrenal insufficiency, œdema of the lungs, Banti's disease, polycythemia, serum therapy, and the surgical treatment of internal diseases, as well as other matters that need not be mentioned in detail. There is an interesting short paragraph on what the author speaks of as the cult of the day—faith healing—that well repays the reading. The book has already been translated into French and German, and translations into Spanish and Chinese are in preparation.

Assuredly, therefore, in its new edition, the volume maintains and strengthens the commanding position universally accorded it; it remains, as it always has been, an extremely personal book, not only reflecting the personality and knowledge of medicine of its author, but also exemplifying the depth and breadth of his learning aside from medicine; it is a record of illuminating clinical descriptions based upon wide experience and careful and discerning sifting of large numbers of cases; and it is fully abreast of the times, since no important advance has escaped the discriminating pen of its accomplished author.

A. K.

A PRACTICAL TREATISE ON DISEASES OF THE SKIN, FOR THE USE OF STUDENTS AND PRACTITIONERS. By JAMES NEVINS HYDE, A.M., M.D., Professor of Dermatology in Rush Medical College, Chicago. Eighth and revised edition, illustrated with 223 engravings and 58 plates in colors and monochrome. Philadelphia and New York: Lea & Febiger, 1909.

THE volume in hand is a large, imposing, handsome octavo, numbering over eleven hundred pages, and may be said to cover the field of cutaneous diseases in an exhaustive manner. From the first edition to that now published the growth and improvement have been progressive, a very complete book being the result. A careful perusal of the pages shows that the work of revision has been satisfactorily and conscientiously performed. It is noted that diseases of warm countries and the tropics have been considered in a separate chapter, this grouping, however, bringing together, it need hardly be stated, diseases more or less diverse in nature. New articles have been written on prurigo nodularis, certain forms of erythema, "the fourth disease," paraffin prosthesis, osteoma and calcification of the skin, meralgia paræsthetica, acrodermatitis pustulosa hiemalis, lichen spinulosus, keratolysis exfoliativa congenita, lipoma, Fordyce's disease, causalgia, leukæmia and pseudoleukæmia cutis, tinea ciliorum, and a few other diseases. It may be said that the volume contains such a wealth of material, from whatever standpoint it may be viewed, that it constitutes a most valuable work of reference, to which one may turn with assurance that the subject, no matter how obscure or rare, will be at least touched upon if not fully elaborated. The references throughout are copious and include the literature of many countries, while the illustrations are both numerous and, as a whole, satisfactory. Many of the photographs portray the lesions and other features of the disease admirably (as, for example, "gangosa," pityriasis rubra pilaris, dermatitis venenata, and circinate erythema multiforme), and a number of them, moreover, are new cases. In the chapter devoted to general diagnosis occurs an excellent, concise, and distinctly useful table devised for the investigation of cases, aiding and leading up to diagnosis; and the remarks made on general treatment, internal as well as external, are both sound and to the point. Words of caution and advice are uttered concerning the use of arsenic internally which are worthy of being impressed upon general practitioners, many of whom are too prone to prescribe this drug without the indications existing for its administration. There is no drug that is prescribed more recklessly and with more damage than arsenic for diseases of the skin. The sound views on this subject here and there throughout the book must eventually do much to show that this drug, while of distinct value in some cases, is nevertheless in most diseases to be withheld. The nomenclature in the work is that employed by the most prominent dermatologists and teachers,

although many terms either obsolete or but seldom used, it would seem, might have been relegated to footnotes rather than be made conspicuous in the body of the text. Concerning the volume, so well and favorably known as it has long been, it is unnecessary to say more. It must for many years remain a standard and useful work, and one that the general practitioner especially should possess. As a book for the student, it might perhaps be criticized as being both too voluminous, as well as too elaborate in scope and detail. Author and publisher alike are to be congratulated for such a valuable contribution to literature. L. A. D.

THE PRINCIPLES AND PRACTICE OF MEDICINE. By ARTHUR R. EDWARDS, A.M., M.D., Professor of the Principles and Practice of Medicine and of Clinical Medicine in the Northwestern University Medical School, Chicago. Second edition; pp. 1257; 121 illustrations. Philadelphia and New York: Lea & Febiger, 1909.

ABOUT two years ago, in commenting upon the publication of the first edition of Edwards' *Practice of Medicine*, we took occasion to commend the book to the attention of students, practitioners, and teachers, in the belief that when tried it would not be found wanting, but, on the contrary, always of service. This opinion has found abundant confirmation, which is especially well exemplified in the early demand for a second edition. The book has been really revised, not merely reprinted. As stated by the author, particular attention has been given to therapeutic details, numerous new preparations and modified names and dosages, particularly for children, being explicitly specified. There are practically new chapters on the arrhythmias and other cardiac neuroses, tropical splenomegaly, and various other tropical affections. Due consideration has been given Flexner and Jobling's antimeningococcic serum, Strong's work on amœbic dysentery, blood cultures in typhoid and other bacteremias, the "carriers of infection," epidemic meningitis and poliomyelitis, tuberculosis, the spirochete of syphilis, etc.

Throughout the book, as in the first edition, the causative pathology has been blended with the consecutive clinical features of disease, reasons have been given for facts, exceptions have been subordinated to what is usually found at the bedside, and the allurements of typical clinical pictures and dogmatic generalizations have been avoided, because, as stated by the author, they hold neither in practice nor at the bedside. The volume contains an unusual wealth of well-arranged and well-digested clinical and other facts, perhaps more than may be found in any other book of its kind; much attention has been devoted to diagnosis and differ-

ential diagnosis—which undoubtedly will be appreciated by the harassed busy practitioner; and the details of treatment, based, in large part, upon physiological principles, are unusually full and explicit, enabling the physician to resort to them in practice with assurance of a happy outcome. The book unquestionably is a credit to its author; and in the revised edition is an improvement upon an originally excellent volume. A. K.

DIAGNOSTIC METHODS. By RALPH W. WEBSTER, M.D., Ph.D., Assistant Professor of Pharmacological Therapeutics and Instructor in Medicine in Rush Medical College, University of Chicago. Pp. 641; 37 colored plates and 164 other illustrations. Philadelphia: P. Blakiston's Son & Co., 1909.

THE wide field of work suggested by the title given to Dr. Webster's book has been ably and for the most part thoroughly covered by the author. A glance at the table of contents will show what a systematic and comprehensive compilation one has in his hands. "Compilation" is hardly the correct word, since we learn in the author's preface and throughout the book that many of the methods described have been mentioned by him because of his personal acquaintance with them in his laboratory. Insistence is laid throughout on the macroscopic and unstained examination of a specimen, and on the correlation of the laboratory findings with the clinical aspects of the case. The book comprises laboratory examinations of sputum, of oral, nasal, and conjunctival secretions, of gastric contents, of feces. A special chapter is devoted to parasites, and is admirably illustrated by many good figures. The detail which the author has devoted to his work may be judged of when it is known that 200 pages alone are needed for the urinary examination. The rest of the book describes examinations of secretions of the genital organs, examination of the blood, transudates, and exudates, and secretions of the mammary glands. In a work of this magnitude it is impossible to avoid omissions of minor importance. However, certain well-known tests have been overlooked, or is it because the author has found them valueless? We refer, among others, to Lange's test for acetone, Rivalta's test for differentiating between exudates and transudates, dimethylamidobenzaldehyde test for urobilin, some test for hydrobilirubin in the feces, and Hopkins' method of estimating uric acid. It is, of course, an impossibility to obtain a book dealing with clinical diagnostic methods in which one can find everything he wishes, and for this reason Dr. Webster's sins of omission should not be too severely judged.

Certain tendencies of the author to overload the book with unnecessary facts are shown on page 18, page 45, and page 504. In the first

instance, under the description of the tubercle bacillus, reference is made to the diagnostic use of the tuberculin reaction; in the second instance a histological consideration of the stomach is given, while in the last instance attention is directed to therapeutics and prognosis. A small error is noted on page 49, where an Ewald meal is said to consist of a piece of bread and a cup of tea, and in the next sentence is the statement that this will represent in approximate figures "36 grams of wheat bread and 400 c.c. of water." An ordinary cup is incapable of holding more than 180 c.c. Regret is here expressed that the author's intention to limit the bibliography was adhered to, as some of the descriptions of methods are unclear and too sparse to be of much use. The plates are uniformly good, but exceptions must be taken to three: Plate IV shows no browning of the body of the bacillus as described in the text, while Plates XXI and XXII are so nearly alike that their purpose is not apparent. The above points of criticism will undoubtedly be eliminated in succeeding editions which are sure to follow. The book is to be heartily recommended. E. H. G.

PAIN: ITS CAUSATION AND DIAGNOSTIC SIGNIFICANCE IN INTERNAL DISEASES. By RUDOLPH SCHMIDT, M.D., Assistant in the Clinic of Hofrath von Neusser in Vienna. Translated and edited by KARL M. VOGEL, M.D., Instructor in Pathology, and HANS ZINSSER, M.D., Instructor in Bacteriology, in the College of Physicians and Surgeons, Columbia University, New York. Pp. 326. Philadelphia and London: J. B. Lippincott Co., 1908.

THE forelying manual professes to be a systematic analysis of pain. Its scope is obvious from the chapter headings, which comprise: the sensation of pain; the functional modification of pain, including the influence of position, motion, pressure, food, drugs, chemicals, and organic function; topography, in its relation to pain; the quality and time occurrence of pain; and painful sensations referable to the nervous system, the organs of locomotion, the digestive system, the urinary system, the respiratory system, and the circulatory system; to which the translators have added a chapter on the results of Head's studies of cutaneous tenderness in visceral disease. It is immediately apparent that to the accomplishment of his task the author has brought not only his long experience, but also a critical mind and a philosophical temperament. The result is a book of much merit and unusual interest, and this, too, in spite of the elusiveness of the ultimate nature of painful sensations and the subjectiveness and psychological aspects of pain as encountered by the physician. The majority of physicians doubtless will read with most intentness the chapter of 125 pages on painful sensations in diseases of the

digestive system, wherein are recounted the varieties and modifications of pain in the gastralgias, gastric ulcer and carcinoma, pyloric spasm, appendicitis, lead colic, intestinal colic, disease of the gall-bladder, etc. Perhaps too much is made of a single subjective symptom, but the author delivers his message rather convincingly. At all events the book well repays the perusal. A. K.

PRACTICAL POINTS IN ANESTHESIA. BY FREDERICK-EMIL NEFF, M.D., New York City. Pp. 45.

BLOOD-EXAMINATIONS IN SURGICAL DIAGNOSIS: A PRACTICAL STUDY OF ITS SCOPE AND TECHNIQUE. By IRA S. WILE, M.S., M.D., New York City. Pp. 158; with 35 figures and 2 colored plates.

700 SURGICAL SUGGESTIONS. BY WALTER M. BRICKNER, M.D., ELI MOSCHOWITZ, M.D., and HAROLD HAYS, M.D., New York City. Third series; Pp. 150. New York: Surgical Publishing Company, 1909.

DR. NEFF'S *Practical Points in Anesthesia* gives some valuable points in the administration of chloroform and anesthol, the latter being a combination of chloroform, ether, and ethyl chloride. The value of the book would be greatly increased if the practical points in the administration of ether anesthesia were included. From the title of the book one would suppose that they would be given. The routine administration of morphine before all anesthesias is to be generally condemned. The author, however, advises it even before giving chloroform and anesthol. We would suggest that the title of the book be changed to *Practical Points in Chloroform and Anesthol Anesthesia.* The book altogether is a valuable one, and if its reading will make anesthetists, and doctors generally, more careful in the administration of all anesthetics, and particularly chloroform, it will have accomplished a noteworthy object.

Dr. Wile's *Blood Examinations in Surgical Diagnosis* presents in a compact form the technique of blood examinations in general, and treats more particularly of blood changes in surgical and gynecological conditions. The immense literature on hematology has been carefully collected and is presented briefly in reference to its practical importance and value in surgical diagnosis and prognosis. Of special excellence are the chapters on "the blood in obstetric and gynecological conditions," and "blood examinations in surgical operations." The book is very practical, and to the student of general surgery and surgical diagnosis it adds a most valuable chapter to his knowledge of the subject.

700 Surgical Suggestions has reached its third edition, it being first presented in 1906 as *500 Surgical Suggestions.* The text consists

entirely of practical suggestions arranged systematically to the various regions of the body. In scope the work is very complete; and it contains many practical points for the student and practitioner which are difficult to locate in general text-books on surgery. The book may be recommended thoroughly for the purpose the authors had in view in preparing it. J. A. K.

Epoch-making Contributions to Medicine, Surgery, and the Allied Sciences. Collected by C. N. B. Camac, M.D., of New York. Pp. 435. Philadelphia and London: W. B. Saunders Co., 1909.

Dr. Camac has placed us under considerable obligation by collecting in a single volume eight of the important contributions that have significantly influenced medical thought and practice—really epoch-making contributions. These are Antisepsis, by Lord Lister; The Circulation of the Blood, by William Harvey; Percussion of the Chest, by Leopold Auenbrugger; Auscultation and the Stethoscope, by R. T. H. Laënnec; Vaccination against Smallpox, by Edward Jenner; Anesthesia, by William T. G. Morton; and Puerperal Fever, by Oliver Wendell Holmes. Dr. Camac very truthfully points out that we may turn for inspiration and knowledge to these early writings, stating that "in many instances subsequent writers have merely paraphrased the statements of the original observers; indeed, one may go farther and say that some of the errors of today are the result of disregarding or misquoting the facts clearly set forth in these original treatises." The book should be read by every member of the profession, not only by those grown old in the service, but by the youngsters who often need, and assuredly thus will secure, much needed stimulation to careful and painstaking work. A. K.

Progressive Medicine. A Quarterly Digest of Advances, Discoveries, and Improvements in the Medical and Surgical Sciences. Edited by Hobart Amory Hare, M.D., Professor of Materia Medica and Therapeutics in the Jefferson Medical College. Assisted by H. R. M. Landis, M.D., Demonstrator of Clinical Medicine in the Jefferson Medical College, Philadelphia. Vol. III, 1909; pp. 336. Philadelphia and New York: Lea & Febiger, 1909.

Volume III of *Progressive Medicine* for 1909 opens with an instructive account, comprising 106 pages, of diseases of the thorax

and its viscera, including the heart, lungs, and bloodvessels, by William Ewart. Special mention is made of tuberculosis, the respiratory gases and aërotherapeutics, the physical signs and examination of the chest, pleural effusion, common colds, asthma, physical examination of the heart, heart strain and overstrain, arteriosclerosis, the arterial wall and blood-pressure readings, etc. William Gottheil devotes 50 pages to dermatology and syphilis. Edward P. Davis devotes 124 pages to obstetrics, special mention being made of disorders and complications of the pregnant state, the toxemias of pregnancy, placenta prævia, labor and its complications and their management, obstetrical surgery, and the management of the puerperal period. William G. Spiller devotes 49 pages to diseases of the nervous system, noting in particular tumors of the brain, aphasia, apoplexy, meningitis, cerebral syphilis, tabes dorsalis, poliomyelitis, neuritis, epilepsy, etc. The publication continues to merit the professional favor bestowed upon preceding issues.

A. K.

THE PRINCIPLES OF HYGIENE AS APPLIED TO TROPICAL AND SUBTROPICAL CLIMATES AND THE PRINCIPLES OF PERSONAL HYGIENE IN THEM AS APPLIED TO EUROPEANS. By W J. R. SIMPSON, M.D., F.R.C.P., D.P.H., formerly Health Officer of Calcutta; Lecturer on Tropical Hygiene at the London School of Tropical Medicine; and Professor of Hygiene, King's College, London. Pp. 396; 98 illustrations. London: John Bale Sons & Daniels Son, 1908.

THIS book deals essentially with the subject of hygiene as adapted to the requirements of the tropical British possessions. The dangers of the tropics and the differences in conditions there from those of the temperate zone are clearly pointed out. Practical, simple, and efficient advice is given how to escape these dangers and how to meet these differences. The book represents the application of sound, modern hygienic principles to the conditions of tropical countries. Emphasis is laid upon personal and domestic hygiene, while such subjects as the examination of water, communicable diseases, and sewage disposal are adequately treated. Laboratory methods are given in a concise and clear manner, and those that are both practical and useful in the tropics are included. However, there is for the most part a freedom of technical detail, and the clear simple style makes the book of value to a reader not possessing a highly technical training. The paragraphing is well arranged and makes the book suitable for purposes of reference. The many illustrations serve mainly to point out the worse features of the poor sanitation in the tropics, and to give plans for proper sanitary construction, as has been found especially useful in hot climates. The writer has gathered much useful information during his sojourn in the tropics, and the

book contains many practical suggestions. The details of the text seem to be correct and in accord with the latest principles of hygiene. The book should be a valuable possession for either a medical man or a layman visiting a tropical or subtropical country.

G. C. R.

MANUAL OF THE DISEASES OF THE EYE FOR STUDENTS AND GENERAL PRACTITIONERS. By CHARLES H. MAY, M.D., Attending Ophthalmic Surgeon to the Mt. Sinai Hospital, New York; Consulting Ophthalmologist to the French Hospital, to the Gouverneur Hospital, and to the Italian Hospital, New York. Sixth edition; pp. 600; 362 original illustrations, including 22 plates, with 62 colored figures. New York: William Wood & Co., 1909.

THIS book has passed through six American editions and the same number of reprints in nine years. Three editions have also appeared in England, two in German, the same number in Italian, Dutch, and Spanish, and a translation also in French and Japanese. This is surely a remarkable history and most convincing evidence that the work has fulfilled the purpose which the author proposed to himself in the preface to the first edition—"to present a concise, practical, and systematic Manual of the Diseases of the Eye, intended for the student and general practitioner of medicine." This sixth edition, while not increased in size, has been brought up to date. Transillumination, the conjunctival tuberculin reaction, decompression, cyclodialysis, exsection of the sympathetic, are among the newer subjects treated. The figures and plates with which the book is richly illustrated are a valuable addition. Some of the colored plates, though generally quite satisfactory, leave something to be desired in fidelity to natural coloring.

T. B. S.

THE SURGERY OF THE EAR. BY SAMUEL J. KOPETZKY, M.D., Attending Otologist to the New York City Children's Hospitals and Schools. Pp. 368; 75 Illustrations. New York: Rebman Company, 1908.

THE operative surgery of the ear has advanced so much in recent years that its literature has assumed an enormous bulk. Dr. Kopetzky is entitled to the gratitude of the American profession for the able manner in which he has collated the literature of operative otology and the lucidity with which he describes aural operations. This book presents unmistakable evidence of large experience and good practical work on the part of its author. One of its most useful features is the complete bibliographies which it contains of the various operative procedures. The book is well worthy of a place in the library of any one interested in the subject of otology.

F. R. P.

PROGRESS

OF

MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

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Polycystic Disease of the Kidneys.—COOMBS (*Quart. Jour. Med.*, 1909, iii, 30) reports a case of cystic disease of the right kidney, absence of the left kidney, multiple hemorrhages, and death from uremia. With a view to determining whether there is any relation between the amount of healthy kidney substance and the onset of symptoms indicating systemic poisoning, such as are associated with chronic nephritis, he has studied the notes of 45 cases of cystic kidney; 32 showed advanced cystic disease, and of these, 18 had fatal symptoms of uremia, while among 12 moderate cases there were 4 such deaths. In 9 others death was due to cardiovascular disease. Circulatory changes due to heightened arterial pressure, œdema, and capillary hemorrhages into the skin or mucous membranes may be present in both cystic kidney and chronic nephritis. There is, thus, clinically, a close analogy between these two conditions which are pathologically marked by destruction of renal tissues.

Rheumatic Arthritis and "Scarlatinal Rheumatism."—POYNTON (*Quart. Jour. Med.*, 1909, iii, 15) has traced the after histories of 25 cases of rheumatism in childhood directly associated with scarlet fever, and finds that the clinical symptoms are identical with those of acute rheumatism. One case was fatal from pericarditis, and from it a diplostreptococcus was isolated which showed the same characters as the diplococcus found by Poynton and Paine in acute rheumatism. The portal of entrance in the two diseases is apparently identical, for scarlatinal rheumatism begins either soon after an initial sore throat, or in association with a secondary sore throat. The children who suffered from rheumatism during scarlet fever were liable to relapses, in which

multiple arthritis, chorea, or heart disease might light up again. Nodules, purpura, erythemas, and psoriasis are also found in association with both true and scarlatinal rheumatism. Both diseases are benefited by salicylates. In children, scarlatinal as well as rheumatic arthritis is frequently followed by valvular heart disease. In adults in both types, the arthritis is a more prominent feature than the cardiac involvement. Poynton is inclined to believe that more thorough bacteriological investigation will show a close relationship between the streptococci isolated in scarlet fever and Micrococcus rheumaticus.

Remissions and Recovery in Tuberculous Meningitis.—MARTIN (*Bra*; 1909, xxxii, 209) has made a critical study of the reported instances of remissions and of recovery in tuberculous meningitis. Disregarding a number of cases in which the correctness of the diagnosis was probably but not absolutely certain, there were 7 cases in which remissions occurred and in which the clinical condition was proved. In 3 of these instances postmortem examinations showed an old, healed tuberculous meningitis. In 2 the diagnosis was made by the development of tuberculosis in a guinea-pig injected with the spinal fluid. In one the inoculation test was positive, and tubercle bacilli were found in a smear of the spinal fluid. In one case the inoculation test, autopsy, and the presence of a tubercle of the choroid all helped to establish the diagnosis. These patients were all children. The remissions varied from four months to two and one-half years in length. There were 22 cases classed as recoveries in which the diagnosis was certain. In 7 of these tubercle bacilli were demonstrated in the spinal fluid. In 7 cases the inoculation of guinea-pigs was positive. In 3 cases autopsy showed the old tuberculous lesion. In 1 case the bacilli were found in the fluid, and later the old lesion was found at autopsy. In one case both examination of the fluid and inoculation were positive. In three instances the diagnosis was made very certain by the presence of tubercle of the choroid. In none of these cases was there, so far as is known, any recurrence of the meningitis. Martin calls especial attention to the liability of confusion of tuberculous meningitis with "meningism" occurring during the course of infectious diseases and intoxications, and serous meningitis in which there is an exudation of serum into the subarachnoid space, but in which the symptoms are usually less marked. In making the diagnosis of tuberculous meningitis he would rely only on the demonstration or cultivation of tubercle bacilli from the spinal fluid, and on the inoculation test. The presence of a lymphocytosis in the spinal fluid is of less value, for it may occur in other diseases, and some few cases of tuberculous meningitis may show a polynuclear leukocytosis. It is important that a healed lesion of the meninges may form a starting point of a fresh, fatal infection. The treatment in the cases which recovered was so varied that it offers no direct indications.

A Bacteriological Study of Poliomyelitis.—POTPESCHNIGG (*Wien. klin. Woch.*, 1909, xxi, 1334) reports a bacteriological study of 14 cases of recent poliomyelitis. Particular attention was directed to the cerebrospinal fluid obtained by lumbar puncture. In all of the cases

there was constantly present a Gram-positive coccus, usually arranged as a diplococcus, at times as a tetracoccus. The organism was present in small numbers in the fresh fluid. Bouillon tubes to which 1 to 2 c.c. of fluid was added showed a luxuriant growth after one to three days in the incubator. Round, yellowish-white colonies developed on agar plates which were inoculated with the spinal fluid. In these colonies the Gram-positive coccus was present in pure culture. Only once did the organism grow on blood serum. Whether this organism is identical with the others which have been described, whether it is the cause of poliomyelitis, and what its relations are to the meningococcus have not been determined. In one case of poliomyelitis the same diplococcus was cultivated from the blood one day after the onset. A more detailed account of this microörganism will appear later.

The Prognosis and Treatment of Pellagra.—In a general review of the subject, LAVINDER (*Public Health Reports*, 1909, xxiv, No. 37) concludes that the prognosis in all cases is grave as to final and complete recovery. The average mortality, founded on asylum reports is 67 per cent. He believes that the earlier the diagnosis is made and treatment begun, the better the outlook. The chronic type of the disease is the more hopeful. Acute manifestations, fever, mental involvement, nervous disturbances, moist, extensive erythemas, progressive emaciation, especially if accompanied by inveterate diarrhœa, all increase the gravity of the prognosis. The same is true of such complications as malaria, bronchitis, pneumonia, nephritis, intestinal parasites, etc. In considering treatment the most important factor is prophylaxis, and here general hygiene plays the major part. He believes that good corn is wholesome, but points out the difficulty in differentiating it from harmful corn, and urges that it be admitted into the dietary of institutions, such as insane asylums, only with the utmost caution. Among drugs arsenic has given the best results. The newer preparations, atoxyl, first recommended by Babes, arsacitin, and soamin, have been tried, but Lavinder inclines to favor the use of Fowler's solution. He states that the combination of atoxyl with arsenic trioxide, which has been recently advocated by Babes as giving brilliant results, has not proved efficacious in the cases observed by himself.

Tubercle Bacilli in the Blood.—SCHNITTER (*Deut. med. Woch.*, 1909, xxxv, 1566) has examined the blood of tuberculous patients for tubercle bacilli. By means of animal inoculation it has been amply demonstrated that the blood and organs of patients dead of pulmonary tuberculosis or general miliary tuberculosis may, and usually do, contain tubercle bacilli. Schnitter has used the method proposed by Staubli, in which the blood is withdrawn from a vein (1 to 2 c.c.) and immediately placed into ten to fifteen volumes of 3 per cent. acetic acid. The whole is centrifugalized, and smears are made of the sediment and stained in the usual way. Of 34 cases of pulmonary tuberculosis examined in this way, 10 showed tubercle bacilli in the blood. Eight of the positive cases represented severe, advanced stages of the disease, while 2 positive results were obtained in patients in the second stage (according to Gerhardt-Turban). In one case of tuberculosis of the testicles and

bladder, tubercle bacilli were demonstrated in the blood; the lungs gave no evidence of an active process in this case. The diagnostic value of this procedure is largely the aid it offers in the differential diagnosis of general miliary tuberculosis, typhoid fever, and sepsis, as was well proved by one of the author's cases. (After the conclusion of his work the author found certain improvements in technique which appear not to have been thoroughly tested.)

Experimental Parotitis.—HERB (*Arch. Int. Med.*, 1909, iv, 201) isolated a diplococcus from the lung, testicle, cerebrospinal and pericardial fluids, bile, and parotid gland of a man who died having mumps. The organism is similar to that which has been previously isolated from the secretion of Steno's duct, and from the blood in cases of acûte parotitis. The organism grows rather slowly on ordinary media, but more abundantly on a mixture of agar with saliva. A monkey inoculated through Steno's duct with a forty-eight-hour culture developed fever and an acute parotitis lasting six days. A similar reaction was observed in dogs. The pathological lesion was studied at various stages and consists in a non-suppurative parotitis, the infiltration being composed largely of mononuclear cells.

The Occurrence of Altmann's Granules in the White Blood Cells.—In hematology there is much confusion as the relationship of the large lymphocyte to the non-granular antecedents of the myelocytes (myeloblast). Schridde and Naegeli, in particular, have laid great stress on the presence of perinuclear, fuchsinophile granules in the lymphoblast and their absence in the myeloblast, and have sought to differentiate the two cells by the presence or absence of these granules, first demonstrated by Altmann. The staining technique is difficult and in most hands has given unsatisfactory results. BUTTERFIELD, HEINEKE, and MEYER (*Folia Hemitolog.*, 1909, viii, 325) have simplified the technique and have used it in the study of these cells, not only in the bone marrow, but also in the blood in health and disease. As a result of their work they find that Altmann's granules are present not only in the lymphoblast but also in the myeloblast, and they conclude that this method of differentiation is, therefore, valueless.

The Gaseous Exchange in the Lungs in Erythrocytosis.—SENATOR (*Zeit. f. klin. Med.*, 1909, lxvi, 349) has studied the further cases of erythrocytosis, bringing the total number of cases of this disease in which the gaseous exchange in the lungs has been determined up to 8. The reports in the literature show that definite abnormalities were noted in all. The volume of the respired air is increased quite constantly and with this increase there is an augmented output of carbon dioxide and a similar change in the consumption of oxygen. In a third case of erythrocytosis which recently came under Senator's observation the gaseous exchange which was high during the earlier examinations fell to normal in the course of a few weeks. Whether this was due to oxygen inhalations or not remains undetermined. Senator has examined other cases with polycythemia which lacked splenomegaly and were not characteristic of Vaquez's disease, and in none of these did the gaseous exchange of the lungs depart from the

normal. In the absence of polycythemia one finds an increased gaseous exchange during the digestion of a hearty meal, without a coincident increase in the volume of the respired air. Similarly, Senator finds that artificially induced hyperemia of the lungs (by the Saugmaske) leads to an augmentation of the carbon dioxide and oxygen. In one of the latter cases the results were quite unexpected, since the patient suffered from chlorosis (the hemoglobin being 27 per cent.). No increase in the volume of the respired air occurred. Senator believes that the increase of the gaseous exchange in Vaquez's disease cannot be attributed to any defect in the hemoglobin, since in many cases this has been found to be normal, and also because a decrease of the oxygen capacity of the blood could not explain the increased gaseous exchange, although it might account for the polycythemia and the activity of the bone marrow. Senator finds a striking similarity in the gaseous exchange of healthy individuals during digestion and in patients with erythrocytosis examined while fasting.

SURGERY.

UNDER THE CHARGE OF

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Rectal Anesthesia.—LEGUEU and VERLIAC (*Archiv. gén. de chir.*, 1909, lv, 551 to 661) describe a method of administering ether per rectum, and say that the essential points to be observed in order to avoid accidents are: (1) During the administration the ether should be kept below the boiling point by immersing the flask containing it in a wet bath at 33°. (2) The anesthetic vapor should be introduced slowly, in order that the normal intestinal gas should be admixed with a progressively increasing quantity of the ether, and that the anesthetic should not be substituted for the intestinal gas. All the ether introduced should be charged with air. When the operation is completed, the rectal tube should be disconnected and as much as possible of the ether expelled with the aid of abdominal massage. The technique of rectal anesthesia is far from being perfect at the present time. The employment of bromide of ethyl as a preliminary inhalation, of ether not heated, and of oxygen as a vehicle, has permitted the avoidance of the greater number of the accidents imputed to the older technique. Prudently conducted, rectal narcosis is not more dangerous than narcosis by inhalation. It has some contra-indications (bad or doubtful conditions of the intestine, acute or chronic affections of the abdomen, operations upon the perineum or genital

organs). It does not offer any advantages over the other methods of anesthesia in operations on the extremities, but it does offer some indisputable advantages in operations upon the face, the buccal cavity, the neck (the field of operation being left free to the operator who is not disturbed or contaminated by the anesthetic), and upon the thorax (condensed ether vapors are injurious to the lungs). It is a method to be used only exceptionally.

Gunshot Wounds of the Kidneys.—Clement (*Ann. d mal. d. org. gén.-urin.*, 1909, ii, 1281) says that the tampon should be employed in those cases in which there are no immediate signs of injury of the kidney or in which these signs come on slowly. In these cases we may consider that the lesions are benign and the hemostasis due to spontaneous clotting. Bleeding may, however, occur later in these cases, from the withdrawal of the tampon or the separation of a slough. Of 6 cases of nephrectomy for gun-shot wounds of the kidney, 3 recovered and 3 died. Anuria is due in the greater number of cases to a reflex inhibition, produced by the trauma of the injured kidney upon its fellow, and signifies only an important lesion of the renal parenchyma. The co-existence of a wound of entrance in the lumbar region is an indication for an exploratory incision. In two of the fatal nephrectomies, one case was complicated by grave stomach lesions, the other by similar lesions and by a wound of the pancreas, all complications which could have produced a fatal termination. In the 3 cases which recovered, the signs were distinct enough to permit early diagnosis and operation, before the hemorrhage had become serious or infection had developed. Early operation is very important. There is less danger from too early operation than from not operating early enough.

Cure of Prolapse of the Rectum Obtained by Tampon.—Sick (*Zentralbl. f. Chir.*, 1909, xxxvi, 1225) says that Ekehorn has described a simple but rational operative method of treatment for prolapse of the rectum in children. This consists in passing a needle carrying a silk suture alongside the sacrum into the lumen of the rectum and under the guidance of the index finger of the other hand, bringing it out of the anus. It is then passed backward from the inside of the rectum outward alongside the sacrum. By this suture the rectum and the included connective tissue in the sacrococcygeal concavity are securely fixed against the bone. The suture is removed in about two weeks, the resulting cicatricial tissue making the fixation permanent, Ekehorn secured good healing in 4 cases in children. Previous to the publication of Ekehorn's work, Sick had tried, with good success, to obtain the same result by simply tamponing the retrorectal space. A longitudinal incision is made in the raphé between the end of the coccyx and the circular fibers of the sphincter ani muscle, where there are no vessels, nerves, or muscular fibers to be injured. The superficial fascia and the deep pelvic fascia are divided, and the loose connective tissue behind the rectum exposed. The rectum is then freed on its posterior wall by a suitable instrument, as high as the promontory of the sacrum. In the cavity thus made, a strip of iodoform gauze of four to six thicknesses is laid, and the small external wound protected from the anus

by an adhesive plaster or collodion dressing. This method is applicable to those cases which recur after the usual treatment by recumbency in bed and keeping the buttocks together by adhesive plaster. It is simple and less dangerous than Ekehorn's suspension method, and gives more promise of a permanent cure, because of the greater cicatricial adhesion developed.

The Operative Reduction of Old Dislocations of the Elbow.—DOLLINGER (*Deut. Zeit. f. Chir.*, 1909, c, 38) says that he has treated thirty-four old dislocations of the elbow. In the first cases he attempted reduction without operation. In no case did he succeed when the dislocation was more than three weeks old. The operations which followed showed such anatomical changes as to render reduction impossible without exposure of the body ends of the joint. He now operates on any case that is more than three weeks old. In the beginning he tried to preserve the lateral ligaments intact. The joint was exposed externally, the trochlea and capitellum were freed from the surrounding tissues, and the reduction attempted by hyperextension, pulling, and flexion. Of thirteen cases in which this method was tried, only three were successful. In the remaining cases it was necessary to detach the lateral ligament from the epicondyle or to detach the epicondyle with the ligament. In all cases the ligament had become shortened. The force necessary to reduce the dislocation, without division of the external ligament, caused injury to the cartilaginous ends, and this influenced the function later. These considerations led to the adoption of the following method: The joint is exposed by an incision, 12 to 15 cm. long, extending on the outer side of the arm along the lateral intermuscular septum to the epicondyle, which is detached with the lateral ligament by a chisel. The ends of the bones are freed from the surrounding tissues only so far as is necessary to permit the bending inward of the forearm until it lies alongside the arm, when the dislocated joint ends can be pushed together out of the wound and freely inspected. If it develops that the bone ends have undergone such changes that physiological function is impossible after reduction, a resection is done. Of the 34 cases, in 14 resection was considered necessary and 20 were considered suitable for reduction. Even in those cases which were reduced, the articular fossa of the olecranon was filled with fat, remains of capsule, and detached fragments of bone, all of which had become bound together by cicatricial tissue. All these tissues were separated and removed, the joint cartilage being preserved. When the bones are thus prepared the reduction is accomplished not by a pull, but after stretching the internal ligament distalward until the inner articulating surface of the olecranon is brought against the inner articulating surface of the trochlea. The forearm is then bent outward from its position alongside the arm until the joint surfaces are in their normal relations. The elbow is then flexed to a right angle and the detached external lateral ligament re-attached by suture in its normal place, or, if necessary from shortening of the ligament, somewhat lower. The wound is then closed. Of the 20 cases in which reduction was accomplished, the end results are known in 12. In 2 the joint is completely ankylosed, and in 1 there is slight

motion. These 3 cases were complicated dislocations, and were more suitable for resections than reductions. The remaining cases have movable joints. 2 of them have complete flexion, and extension to 135 degrees. In the 14 cases in which resection was done, only the trochlea and capitellum were removed. The epicondyles were preserved and the lateral ligaments re-attached by suture to their normal points of insertion. Dollinger has reported concerning the later function in 11 cases. In 6 the elbows are stiff, in 5 movable. One patient has complete movement and two have motion of about 85 degrees, and two of from 40 to 50 degrees. These data show the functional end results to be better after reduction than after resection, and they will be better in the future from the employment of the method described here. It follows, therefore, that resection is indicated only when the joint surfaces have undergone such changes that normal function after reduction is impossible.

Treatment of Congenital Dislocations of the Hip.—KÜSTER (*Deut. Zeit. f. Chir.*, 1909. c, 52) reports 10 cases of congenital dislocation of the hip in which reduction was made, one by operation according to Hoffa's method, and 9 by the non-operative method. The steps of the non-operative method as carried out by Küster are as follows: Extension is applied to the limb for a week to stretch the tissues and to bring the femoral head nearer to the acetabulum. Under complete anesthesia the pelvis is held firmly by the hands of an assistant against a firm support underneath. Then by pulling upon the thigh and pressure upon the trochanter the head is brought downward, and if this fails the thigh is brought slowly into extreme abduction. At the same time with the closed fist an assistant presses strongly upon the neck and head of the femur from above. All this is done gradually and without undue roughness. Finally, internal rotation of the femur is made slowly and carefully, at times also external rotation. This causes the head to be reduced with a snapping sensation, or it stands so firmly in place that it does not go back into the dislocated position. If a double dislocation exists, the other side is treated in the same way. The thigh is then fixed in the abducted position by a plaster bandage dressing, the legs below the knee not being included in the plaster. Should the *x*-rays show that the head has again become dislocated, the efforts at reduction should be repeated in from ten to fourteen days. The earlier the age at which the dislocation is reduced the more nearly normal are the joint surfaces. The early use of the *x*-rays is very valuable in making the diagnosis and in disclosing the nature of the joint conditions present. One should not become discouraged if a re-dislocation takes place after the efforts at reduction have been made. After repeated attempts a final success may be expected.

Myotomy and Myorrhaphy.—BARDENHEUER (*Deut. Zeit. f. Chir.*, 1909, c, 63) says that lengthening of the muscles which have undergone fibrous degeneration (flexors), combined with shortening of the extensors, gives a better result than the Mikulicz resection of the forearm bones, or the plastic lengthening of the flexors as done by Schramm. In cases of infantile, spastic hemiplegia, the division of the markedly spastically contracted muscles (supplied by the median nerve chiefly),

together with the shortening of the tendons of the simultaneously spastically contracted but weaker muscles (supplied by the musculocutaneous nerve), is more effective than the plastic operation of Hoffa and probably more effective than the Spitz grafting of a portion of the median nerve into the musculocutaneous. In very extensive resections of joints, in which, for instance, at the shoulder, almost half of the humerus has been sacrificed, the muscles surrounding the joint may be removed, so that the defect in the muscles will correspond to that in the bone. This operation would exclude the formation of an extensive wound cavity, would bring the resected surfaces in contact with each other, would prevent the development of a flail joint, secure a movable joint, and render many amputations unnecessary. For the present, Bardenheuer prefers the Lexer transplantation of joints, because of the excellent idea involved and the good results obtained in these operations. The muscle resections may be employed also with resections of the bone in continuity.

A Case of Habitual Dislocation of the Patella.—BUNTS (*Surg., Gyn., and Obstet.*, 1909, lx, 117) reports a case of exaggerated double external dislocation of the patella of an exaggerated type, in which the following operation was done: A linear incision, six inches long, was made on the inner side of the knee, through the skin and subcutaneous tissue, down to the capsule of the joint. A curved incision was then made through the capsule. The cut edges of capsule were overlapped by pulling the inferior margin under the superior margin by mattress sutures, while the patella was shoved forcibly inward. The free edge was then sutured to the lower portion, and the wound closed without drainage. Sterile dressings and a plaster bandage were applied and allowed to remain on for four weeks. Healing by first intention occurred in both knees. The final results have been only partially successful. The patient is able to walk without crutches. The left knee has remained cured up to this time, but the patient has had several attacks of pain in the right knee. The patella is not thrown out of place, but the symptoms are those of a floating cartilage, a condition that is sometimes associated with repeated attacks of dislocation.

Concerning the Search for Perforated Gastric Ulcers.—EWALD (*Zentralbl. f. Chir.*, 1909, xxxvi, 1281) calls attention to the great difficulty sometimes experienced in locating the small openings made by a perforated ulcer of the stomach, when the abdominal walls are tense, the patient in shock, and the small space in which the perforation lies covered with turbid, thin stomach fluid. He says that the perforation occurs almost without exception about 1 to 2 cm. above the pylorus, or the same distance below, and that the opening is usually exposed as soon as the overlying liver is slightly elevated. The whole area in which it may be found is rarely larger than about 4 cm. Ewald has so found it in all cases operated on in the last year. Lieblein found in 223 perforations, only 5 in the middle of the stomach and 12 in the neighborhood of the cardia. The duodenal ulcers perforate much more frequently in the upper transverse portion of the duodenum, and usually close to the pyloric ring. In many men with a sharp pre-

cordial angle, a low lying umbilicus, and flat or sunken abdominal wall, the pylorus will be made visible with difficulty on account of the falciform ligament of the liver. In stout men with emphysematous chests, the pylorus is so far from the epigastric line that the poorly movable pylorus is brought out and the ulcer exposed with great difficulty. For these reasons Ewald now makes the abdominal incision not in the median line, but parallel to it and through the right rectus muscle. The falciform ligament of the liver is then pushed toward the median line. The abdomen is opened first in the lower angle of the incision, the finger is introduced and pushes the falciform ligament toward the median line, and the peritoneum is divided upward. The edge of the liver is lifted upward, when one usually comes directly upon the perforation. The exudate clinging to the pylorus and first portion of the duodenum is then cleared away, and the infiltrated fatty appendages are removed. If the perforation has not been exposed by this time, the posterior surface of the pylorus is made accessible. Only in rare instances will this be necessary.

Direct Blood Transfusion by Means of Paraffin-coated Glass Tubes.—BREWER and LEGGETT (*Surg., Gyn., and Obstet.*, 1909, lx, 293) record the results of some experiments undertaken to find some simple method of direct blood transfusion. End-to-end suture as practised by Carrel and union by the ingenious cannula of Crile have been the methods generally employed in the past. A set of glass tubes was made, some straight with uniform caliber, others bayonet shaped, and still others somewhat tapering in shape and caliber, to be used for transfusion from a large adult artery to the small vein of a child. Each end of the tube is notched, to allow a ligature being applied after the tube is introduced into the lumen of the vessel The tube is sterzilied in an autoclave or by boiling, dropped into boiling paraffin, and the excess of paraffin removed by shaking the tube in the air. The following method of transfusion, as described for No. 1, was performed in all the experiments: The external jugular was exposed in dog No. 1, and the common common carotid in dog No. 2. The proximal extremity of the external jugular of dog No. 1 was temporarily clamped and the vein divided, allowing copious hemorrhage from the distal extremity. When the animal began to show signs of weakness and falling pulse, the distal extremity of the vein was ligated. The two dogs were then placed side by side, the carotid artery of dog No. 2 being double clamped and divided between the two constricted areas. The lumen of the proximal portion of the artery was then expanded by three mosquito forceps, and one extremity of the glass tube introduced and held by a silk ligature placed around the vessel in the notch of the tube. The orifice of the proximal portion of the jugular vein was next expanded in the same manner by means of three mosquito forceps. Before introducing the distal end of the glass tube, the arterial clamp was temporarily released and a few jets of bright arterial blood allowed to pass from the tube. It was then quickly introduced into the lumen of the vein and secured by a second silk ligature. As soon as the temporary clamps were removed, blood could be seen to pass from the artery of the donor to the vein of the donee, causing distinct pulsations in the vein, corre-

sponding to the arterial pulse of the donor. Dog No. 1 was quickly revived by the transfusion, which lasted about six minutes. The vessels were then ligated and the wounds closed and dressed. In thirty-one experiments, only three animals died, each from a too rapid transfusion of the blood, causing overdistention of the right heart. On one occasion only did the blood apparently clot in the tube, and that was undoubtedly due to the very small caliber of the tube used. In one other experiment clotting occurred in the vein near the extremity of the tube, due to an accidental separation of the paraffin coating. The writers believe that this is the simplest method yet proposed, that it can be carried out by any surgeon of ordinary experience without the necessity of previous training. It has been employed in one patient suffering from anemia due to gastric hemorrhage. The blood flowed for nineteen minutes through a medium-sized tube without clotting. The patient rallied well. His hemoglobin rose from 30 to 70 per cent. during the progress of the operation, but the patient died the following day from a recurrence of the hematemesis.

THERAPEUTICS.

UNDER THE CHARGE OF

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The Treatment of Erysipelas by Means of Carbolic Acid and Alcohol.—Judd (*Medical Record*, 1909, vii, 268) reports 82 cases of erysipelas treated by this method during the past eight years. His statistics show 67 complete remissions of symptoms in from twelve hours to four days. There were ten delayed recoveries, and the treatment failed in five of the cases. The technique of the method consists of painting with a swab of cotton the entire surface of the involved area, and extending about half an inch into the surrounding and apparently healthy skin, with a 95 per cent. solution of carbolic acid. This is left until the purplish color of the inflamed area is replaced by a pretty complete whitening of the skin. When large areas are involved, it is advisable that only a portion be painted at a time. The second step consists in going over the whitened areas very thoroughly with a second swab saturated with pure alcohol. The swabbing with the alcohol must be kept up until the whitened area becomes pink. Half an inch of the surrounding sound skin must be included, as the bacteria of erysipelas are found beyond the apparently involved area. This method includes the painting of the hairy scalp, the eyelids, the mucous membrane of the alæ of the nose, and the nipple of the breast, if necessary. Judd has failed to note any bad results from this treatment. He says that there has been no marked toxic action of the carbolic in any case so far observed, although the urine is sometimes darkened and of a characteristic odor. Almost the first result noticed by the patient is a complete cessation of the unendurable itching, burn-

ing, and throbbing. Usually within a few hours nausea, if present, subsides, and within twenty-four hours the temperature sinks to normal. The pulse falls very rapidly from 120 or more to nearly normal, the appetite returns, and, except for the swelling, which remains for one or two days longer, the patient is relieved from his distressing symptoms. Judd says that the most of his failures were in young children, and these cases also showed the greatest number of symptoms of absorption of carbolic acid. He says, furthermore, that this treatment has no permanent injurious action upon delicate skins.

The Action of Potassium Bitartrate.—BURWINKEL (*Med. Klin.*, 1909, xvii, 627) confirms the statement of Eichhorst regarding the diuretic action of potassium bitartrate. He advises it especially in cases of ascites as a result of cirrhosis of the liver. He gives 4 to 5 grams three times a day. In anasarca from failure of compensation in valvular disease, he advises a combination of powdered digitalis and potassium bitartrate.

Chloroform—The Ideal Hemostatic in Pulmonary Hemorrhage.—FISH (*Jour. Amer. Med. Assoc.*, 1909, lii, 1918) reports excellent results from the use of chloroform in 19 cases of pulmonary hemorrhage. Fish states that the routine treatment of hemoptysis has always been uncertain and empirical because of the inaccessibility of the bleeding point. He explains the action of chloroform by a lessening of the heart action, a reduction of the blood pressure, and a diminution of the respiratory movement. Since chloroform produces coagulation of the blood in vitro, he says that it is possible that its hemostatic action may be aided by direct contact of the vapor with the bleeding point. He gives from 2 to 4 c.c. of chloroform by inhalation. Results should occur within five or ten minutes. Subsequently the inhalation of 15 to 20 drops every hour is continued for a few days. At the same time ammonium chloride and small doses of codeine are given. Fish also advises the administration of a teaspoonful of magnesium sulphate three times daily to remove excrementitious matter which, if retained in the blood, would stimulate the vasomotor centre and raise the blood pressure.

The Vaccine Therapy of Enteric Fever.—SEMPLE (*Lancet*, 1909, ii, 1668) concludes from his results with the use of bacterial vaccines in enteric fever that the administration of such vaccine is a practical method of increasing the bacteriotrophic substances in the blood, as evidenced by an increase in the opsonic index of those treated by this method. When appropriate doses are given the method is devoid of all risk, is easily carried out, produces no apparent disturbance in the patient's condition, and does not interfere with any other treatment the physician may deem necessary. It would not be possible to generalize from the few cases which he cites as to whether vaccine treatment has a marked effect in cutting short the fever period. In 6 of the 9 cases treated, well-marked improvement set in after the vaccinations. This improvement was especially marked when autogenous vaccines were administered. In the remaining 3 cases there was an amelioration of the clinical symptoms.

The Favorable Effect of Extract of Cannabis Indica Butyricus in Exophthalmic Goitre.—CRÄMER (*Klin.-ther. Woch.*, 1909, xxiv, 590) calls attention to the recommendation of Sée for the use of the fatty extracts of cannabis indica in the treatment of functional disturbances of the digestive tract and heart. Sée considers that this drug is a very valuable sedative for the gastro-intestinal tract, with the advantage over morphine of not causing nausea or vomiting. Sée also found that it was useful in palpitation of the heart and other heart symptoms due to digestive disturbances. Acting upon Sée's suggestion, Crämer has tried this remedy in a number of patients having gastric symptoms. He found that the best effects were obtained in functional disorders. The symptom of pain was especially benefited. With these results in mind, Crämer was led to try its effects in the treatment of exophthalmic goitre. He relates the histories of several cases so treated, and advocates its use in this condition. Palpitation and tachycardia were improved, the patients began to eat, and they increased in weight and strength. The thyroid diminished in size, together with a diminution of the exophthalmos. Crämer believes that the operative treatment of exophthalmic goitre should only be advocated when all of the various medical procedures fail.

Vasodilators in High Blood Pressure.—MATTHEW (*Quart. Jour. Med.*, 1909, ii, 261) states that it has to be recognized that high blood pressure is not necessarily harmful to the individual. In some cases it is to be considered as purely compensatory; therefore, not every case of high blood pressure should be treated with vasodilators. Undoubtedly there is a tendency for the blood pressure to increase progressively, and our aim should be to prevent this increase from being too sudden and exaggerated. Matthew has studied the action of the vasodilators in patients with hypertension by means of the sphygmomanometer. Nitroglycerin, potassium and sodium nitrites, erythrol tetranitrate, and mannitol hexanitrate all showed a powerful vasodilator action, while cobalto-nitrate of potassium proved inert in this regard. Not all cases of hypertension however, responded with a fall in blood pressure. The nitrites produced little or no vasodilator action in certain cases. Moreover, a rise in blood pressure occurred in some cases of advanced Bright's disease. In heart and kidney disease when there is marked œdema nitrites do not act well. After the œdema has disappeared the usual vasodilator action may reappear. He made observations in many cases of hypertension when symptoms had developed, such as pain, headache, giddiness, epistaxis, etc. He invariably found that such symptoms were alleviated or disappeared entirely with a reduction of pressure amounting to about 30 mm. Hg. Furthermore, he observed that, if this fall could be maintained the symptoms did not reappear, and the patient's general condition improved. Matthew then determined the dose of various vasodilators that would produce and maintain such a reduction. Liquor trinitrine (nitroglycerin), in the dose of 2 minims, lowered the blood pressure 20 to 30 mm. Hg. very temporarily. Following this transient lowering there was an almost immediate gradual rise of the pressure, and in all cases the effect of the drug passed off in thirty minutes. Sodium or potassium

nitrite in doses of 2 grains produced a reduction of just over 30 mm. Hg. This action will last two hours and only after this is it necessary to repeat the dose. No benefit is obtained by increasing the dose, and a less dose will not give the desired result. Erythrol nitrate in the dosage of 0.5 to 1 grain will produce the beneficial reduction, and the effect will last about six hours. Matthew found that erythrol nitrate sometimes caused unpleasant symptoms, and recommends the smaller initial dose as safer. Mannitol nitrate acts similarly to erythrol nitrate in doses of 1 grain. Its maximum effect is attained more slowly than erythrol nitrate, and therefore it is probably safer. Matthew concludes by saying that the useful and suitable dose of a nitrite for each individual can readily be ascertained by noting the effect of the nitrite as to the amount of fall produced and the time the action lasts.

The Absence of Mucus from the Stomach.—SCHALIJ (*Arch. d. mal. d. l'appar. digest.*, 1909, iii, 79) says that the absence of mucus in the gastric secretion aggravates the prognosis of hyperchlorhydria and of ulcer of the stomach. He believes that the mucus has a protective action against the corrosive action of the gastric juice. Schalij, consequently, advocates the use of various bland protective substances to replace the mucus. Among these he mentions olive oil, almond oil, and gelatin. He gives the olive oil or almond oil before meals, starting with 1 c.c. and increasing gradually to 15 c.c. He also advises the ingestion of fatty foods before meals. This method, Schalij says, has the further advantage of decreasing the hyperacidity.

The Treatment of Anemia in Infancy with Citrate of Iron Administered Subcutaneously.—MORSE (*Jour. Amer. Med. Assoc.*, 1909, liii, 107) says that the percentage of hemoglobin in infancy gradually rises from 55 per cent. to 60 per cent. at the end of the first month to 70 per cent. at six months, where it remains during the rest of infancy. The number of red blood corpuscles in infancy varies between 5,500,000 and 6,000,000. Morse believes that this comparative deficiency in hemoglobin is due to the fact that the infant normally receives an insufficient supply of iron in its food and that the reserve of iron present in the liver at birth is not large enough to keep the percentage of hemoglobin at the adult standard. He is of the opinion that the anemias of infancy are usually of a chlorotic type, although probably not a true chlorosis. Therefore, Morse thinks that iron is especially indicated in the anemias of infancy. Because of the difficulty of giving iron by mouth to infants, and since iron is very apt to disturb the digestion, he advocates the subcutaneous administration of iron. For this purpose he has found an aqueous solution of the citrate of iron very serviceable. This can be put up in perles and sterilized, each perle containing a single dose. This solution is absolutely non-irritating, and the injections are never followed by induration or abscess. The injection, however, is sometimes followed by pain lasting from a few minutes to an hour. Morse gives the injection by means of a glass syringe with asbestos packing, fitted with a platinum needle. An ordinary steel needle is corroded by the solution. He gives an average dose of $\frac{3}{4}$ grain every other day. He says that he has used the citrate of iron in

this way in a number of cases—some of the chlorotic type, some mild cases not of this type, and some cases of severe anemia with splenic tumor—with quite satisfactory results, even in the severe cases. In most of the cases the rise in the percentage of hemoglobin has been more rapid than that in the number of red blood corpuscles. Morse gives the detailed histories of five of his cases, and concludes by saying that he believes that the subcutaneous use of iron is often most advantageous. The results are more marked and are obtained more rapidly than when the iron is given by the mouth, and there is much less danger of upsetting the digestion.

Intravenous Suprarenal Preparations in Collapse.—SOHN (*Münch. med. Woch.*, 1909, xxiv, 1221) recommends highly the use of suprarenal preparations in cases of acute cardiovascular collapse. Their action is prompt, and their use may often tide the patient past the danger point. He reports seven cases in which he has had good results from their use. In one of the cases he failed to follow up the remarkable benefit from a single injection, and ascribes the fatal outcome to his failure to continue the injections.

Pneumococcic Vaccine in Pneumonia.—HARRIS (*Brit. Med. Jour.*, 1909, ii, 1530), with regard to the treatment of acute lobar pneumonia by inoculation, concludes that: (1) Successful inoculation for pneumonia is possible; (2) the inoculation does no harm; (3) a vaccine from one or a number of virulent strains should be used; (4) it should be introduced as early as possible; (5) the estimation of the opsonic index is not necessary; (6) the estimation of the observation of the temperature and physical signs is in pneumonia a sufficient guide in gauging the repetition of the dose. Harris believes that infections of the lungs by the pneumococcus which fail to resolve after an acute pneumonia, as well as pneumococcic infections of other areas, ought certainly to be treated with a pneumococcic vaccine. He adds that these cases appear to afford a reasonable prospect of success.

The Treatment of Epilepsy with Calcium Hypophosphite.—CICARELLI (*Il. Polyclin.*, 1909, xvi, 1) records excellent results from the use of calcium hypophosphite in the treatment of a series of 25 cases of epilepsy. He was led to try this method because of the favorable results reported by other Italian physicians in the treatment of epilepsy with lime salts. Cicarelli believes that the phosphorus contained in the calcium hypophosphite has an additional stimulant action upon the nervous tissue of the epileptics. He gives the calcium hypophosphite in doses of from 10 to 15 grains three times a day. Cicarelli advocates this method of treatment either alone or in combination with the bromide method. When given in combination with bromides, the bromides should be given for one week alternating with calcium hypophosphite over a period of two weeks. He considers that the calcium salts are preferable to the alkaline bromides, as bromidism is avoided. In his series of cases patients on calcium hypophosphites were less depressed and their general conditions was better than that of the patients on bromides.

PEDIATRICS.

UNDER THE CHARGE OF
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Spina Bifida Occulta.—Spina bifida occulta is a congenital deformity of the spine characterized by a more or less evident vertebral cleft extending over one or more vertebræ, and without the existence of a tumor. In the dorsal and lumbar regions, where the condition is more common, there is often a profuse overgrowth of hair over the defective arches. The latter is a pathognomonic sign, but may not develop until after puberty. Hip dislocation, clubfoot, partial paralysis of the lower extremities, changes in tactile and thermal sensation, anesthesia, hyperesthesia, and trophic ulcers may be the result of this condition. The deformity is always congenital and usually due to traction of amniotic bands preventing union of the borders of the medullary groove; various theories exist to account for these bands. Symptoms and complications may not arise until the child becomes older. No treatment is indicated if no symptoms or complications exist; if symptoms exist and bands are suspected, they must be searched for carefully and divided under strict aseptic precautions. J. W. SEVER (*Boston Med. and Surg. Jour.*, 1909, clxi, 388) reports 11 such cases, 2 boys and 9 girls; 4 cervical, 1 dorsolumbar, and 6 lumbar cases. Paralyses existed in 4, dislocation of hip in 2, and scoliosis in 2. A careful study reveals them to be the usual varieties of spina bifida occulta. Hypertrichosis was not constant. Not a great deal can be done for the paralytic cases, except to attempt the correction of the deformities.

Fatty Infiltration of the Liver in an Infant Aged Three Months.—C. A. PRATT (*Boston Med. and Surg. Jour.*, 1909, clxi, 395) reports the case of a child, perfectly healthy at birth and remaining so until its eleventh week, when, without any other apparent cause, except the heat, the infant lost its appetite and vomited after almost every feeding. The child was breast-fed until its seventeenth day, the illness of the mother forcing modified milk feeding thereafter. In the course of a journey, non-sterilized milk had to be given, and vomiting became more frequent, the baby becoming more and more ill. Physical examination revealed nothing but a liver markedly increased in size; it extended from the anterior superior spine of the right ilium to the umbilicus. From then on until her death, no matter what form of milk was given, whether human, cow's, modified, or even condensed milk, symptoms of toxemia appeared. At the autopsy the liver was seen to be greatly enlarged, pale yellow, and firm. The microscope showed "no normal liver tissue," fat replacing the protoplasm of the cells. The other organs were reported normal. Pratt believes the excessive amount of fat in the mother's milk (5 per cent.) to have been the cause of the fatty infiltration.

Idioglossia.—Idioglossia is an inability to pronounce certain consonants and substituting other consonants or vowel sounds for them. Hadden was the first to describe this condition in the *Jornal of Medical Science*, 1891. L. G. PARSONS (*Birmingham Medical Review*, 1909, xiv, 9)describes two such cases in brothers, respectively ten and fifteen years of age. Both are bright and intelligent, but rather excitable and nervous. They cannot keep still for any length of time, and concentration of attention is impossible. There is no evidence of mental deficiency in either, and the elder brother writes and copies well; neither stutters. Three other children in the family pronounce words properly. The only consonants the elder boy pronounces correctly are b, d, l, n, t, y, and the younger, l, n, p, t, v, x, y, and even they are not employed correctly in combinations forming words. Concerning the causes, Parsons discards the following theories: (1) That idioglossia is baby language; (2) that it represents atavism or "sport" in language; (3) that it indicates mental deficiency; (4) the theory that it is a congenital deficiency of audition, not amounting to deafness, and an analogous condition to color blindness, is an explanation for some cases, but not for the majority.

The theory explaining all cases is, that it is due to some fault in Broca's area. Of the exact nature of the irregularity we are still in ignorance. Intelligence is not at fault, as in babies where both Broca's area and the frontal lobes are undeveloped. Spontaneous improvement after eight to ten years of age and absence of the defect in adults points to this theory as the correct one. The ultimate prognosis is good and the treatment consists in oral training, best performed with the child in an institution, as the parents, being accustomed to the child's imperfection, cannot do the teaching well.

Influenzal Meningitis.—DAVID DAVIS (*Archiv. Int. Med.*, 1909, iv, 323) reports the cases of twin brothers who died of influenza. They became ill on the fifth day after birth. The cases ran an identical clinical course and terminated fatally on the ninth and eleventh days respectively. There was little or no distinct evidence, clinically, of meningeal involvement. The autopsy on the first child revealed, as prominent lesions, acute purulent leptomeningitis and acute enteritis. From the meningeal exudate and from the peritoneal fluid pure cultures of the bacillus of influenza were obtained. The usual atria of infection—nasal and tympanic cavities, lungs, bronchi, and throat—were normal. Omphalitis was not present. The children were breast-fed and were healthy until the fifth day, when the bowel movements became green and contained mucus and curds. Both children then became drowsy and refused the breast, and later developed attacks in which they became cold and cyanotic. There was no rigidity, and no symptoms of meningeal irritation developed until the ninth day, when there were general twitchings, but no actual convulsions. A comatose condition and marked cyanosis preceded death, which occurred in one child on the fourth day of the disease, and in the other on the seventh day. The temperature during the disease ranged from 101° F. to 104.5° F. Neither the mother nor any of the family were afflicted with colds at this time. The autopsy confirmed the intestinal involvement and showed a mild peritonitis. The intestinal tract was the

suspected portal of entry, and the facts suggest a similar infection of common origin in the two cases, a common symptom in reported cases being diarrhœa. The meningeal exudate was greenish yellow, highly purulent, and friable, and was more copious and widespread than is usual in the other forms of meningitis. This type also contains a very large number of bacilli in the exudate, both inside and outside cells. Adams found 21 cases in which the as isolated from the meningeal exudate in pure culture. Besides this, a number of cases are reported in which there occurred a mixed infection of the bacillus of influenza with a gram-positive diplococcus. The typical bacillus of influenza grows only on hemoglobin media, and profusely so on pigeon's-blood agar.

Acute Hydrops of the Gall-bladder in Scarlet Fever.—MONTENBRUCK (*Deut. med. Woch.*, 1909, No. 24, 1065) reports the case of a boy, aged five years, who was taken suddenly ill with the characteristic high temperature and eruption of scarlet fever. Two days after the onset he developed violent vomiting and severe pains in the upper part of the abdomen which lasted, off and on, for about eight days. The vomiting then ceased, but the abdomen remained very tender to pressure. During this time there was a constant fever, ranging between 102° F. and 104° F. On the eleventh day the child was admitted to the hospital with the diagnosis of abscess of the liver. There was distention and rigidity of the abdomen and extreme tenderness over the lower border of the liver and the gall-bladder region. As the child screamed with pain, and the abdominal condition seemed to demand it, an exploratory incision was made over the gall-bladder region, under chloroform anesthesia.

An immensely distended gall-bladder, the size of three fists, presented. It was freely movable and on tapping it was found to have held 250 c.c. of greenish, sterile bile. No stone was found. The gall-bladder was drained for a short time through one end of the incision, the sinus then closed, and the child made an uninterrupted recovery. During the operation a fine desquamation was noticed over the body. The urine remained normal throughout.

Chronic Rheumatoid Polyarthritis.—O. J. KAUFFMANN (*Brit. Jour. Children's Dis.*, May, 1909) reports the case of a girl, aged eight years, who showed enlargement of the nearly all the joints in the body. The joints were fusiform in shape, there was no pain or tenderness on pressure, and no grating. All had a certain amount of passive motion except the right hip-joint, which was ankylosed from an osteophytic growth. The joints were elastic and soft, but there was great wasting of the muscles. The spleen was enlarged, as were the superficial lymph glands all over the body, but they were not tender.

Slight bronchopneumonic symptoms in the lungs soon disappeared. The Calmette ophthalmo-reaction was negative twice. The temperature ranged from 99° F. to 101° F. The child had had three attacks of pneumonia—at one, two, and three years of age; rötheln at four years, followed by pains in the legs but no joint swelling. In the sixth year the joints began to enlarge, with wasting of the muscles, and the

condition had progressed. Treatment was useless. Kauffmann suggests that rheumatoid arthritis in children may be due to a chronic pneumonic condition in the lungs, as shown by the repeated attacks of pneumonia in this case.

OBSTETRICS.

UNDER THE CHARGE OF
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Menstrual Bleeding during Pregnancy.—VOGT (*Zentralbl. f. Gyn.*, 1909, No. 36) has examined the literature of menstruation during pregnancy, finding a record, in 100 cases, of undoubted periodical bleeding in eight. He reports a case in which the patient was a twin child well developed in childhood, having its usual diseases. Menstruation began about the twelfth year and soon became regular, lasting from five to eight days without pain. There was no other discharge. Twenty-two years before coming under observation the patient had her first child normally. When the patient came under observation she was found to be undoubtedly pregnant, as the fetal heart sounds were strong and regular and could be plainly heard. She stated that up to the sixth month of her pregnancy she had menstruated regularly. The pelvis was normal and internal examination revealed nothing abnormal about the genital organs. Other sisters had exhibited during pregnancy the same persistence of menstruation.

Syphilis of the Kidney Complicating Pregnancy and the Puerperal Period.—HIRSCH (*Zentralbl. f. Gyn.*, 1909, No. 35) reports the case of a patient aged thirty years, anemic and highly nervous, who had had three pregnancies, during two of which she suffered from œdema with swollen feet and symptoms of toxemia. In her fourth labor the amniotic liquid was unusually abundant; the patient had a chill during labor with a pulse of 120, and the child was dead-born. The fœtus had hydrocephalus, enlarged spleen with abundant deposit of fibrous tissue in the capsule, ascites, enlarged thymus, and enlarged liver. There were no signs of syphilis in the skeleton. The mother's puerperal period was free from fever, but the secretion of urine was scanty and contained albumin—4 per cent. The urinary bladder contained abundant round epithelium, recognized as coming from the kidney, in groups and in tubes. There were no casts and no leukocytes. Repeated examination found the same thing. On examining the mother, she was found to have grown steadily worse since her confinement. There was leukodema of the upper portion of the body, especially pronounced in the upper thoracic region, axilla, and lymphatic glands. There was pronounced caries of the teeth, and linear scars on the reddish mucous membrane of the lip; on the right tibia there was an exostosis; over both shins there was slight œdema. The history showed that the patient had never made a complete recovery since her third labor; although her appetite had

been good, she had never gained in weight, but had distinctly lost. Pain was often complained of in the region of the right kidney, and the feet often swelled, and all of these symptoms were exaggerated during the last pregnancy. The diagnosis was made, from the examination of the fœtus and the mother, that syphilis in the right kidney complicated pregnancy and the puerperal period. Iodide of potassium was given freely. The quantity of urine increased, albumin disappeared, kidney epithelium grew less, and the pain in the region of the kidney much diminished. The patient increased in weight, the urine becoming normal. Some time after the patient was first seen, she was again examined and symptoms of syphilitic involvement of the nervous system were present. The patient improved markedly under the treatment, although her general condition was not good.

Twin Pregnancy with Abnormal Attachment of the Ova.—LEO (*Zentrbl. f. Gyn.*, 1909, No. 36) reports the case of a woman in her second pregnancy, aged thirty-one years, who had considerable persistent abdominal pain during the pregnancy. The uterus was anteflexed, enlarged, and pregnant about the second month. There was considerable distention and tenesmus of the abdomen. Symptoms of abdominal irritation continued, and a diagnosis of ectopic gestation was made. On opening the abdomen, ruptured tubal gestation was found and intra-uterine pregnancy advanced about three months. The patient's recovery was complicated eight days after operation by slight hemorrhage from the uterus, which subsided with rest and very simple remedies.

Peritonitis Following the Escape of Amniotic Liquid into the Abdomen During Cesarean Section.—VEIT (*Zntrlbl. f. Gyn.*, 1909, No. 32) reports a case of Cesarean section performed upon a patient, who had been for a long time in labor. An effort was made to clamp the peritoneum of the lower uterine segment to the abdominal wall by forceps. The child was removed by version, the clamp became loosened, and amniotic liquid, containing bacteria, gained access to the peritoneal cavity. Peritonitis developed, with the presence of a dark, tenacious exudate in the peritoneum. An effort was made to drain the abdomen by section and the use of a glass tube. The effort was unsuccessful, the patient dying. Autopsy showed degeneration of the heart muscle, with extensive poisoning of the peritoneum with Bacillus coli communis.

Two Cases of Cervical Cesarean Section.—KÜSTNER (*Zntrlbl. f. Gyn.*, 1909, No. 36) reports two cases in which he performed cervical Cesarean section. The first patient had had three labors, the last ending in embryotomy because of a tumor in the pelvis. During the fourth labor she was repeatedly examined outside the hospital, and as it seemed impossible to deliver her, she was brought into the wards. On examination, a pelvic tumor was found, rendering delivery impossible. An effort to dislodge the tumor failed, and the abdomen was opened in the median line. The uterus was opened outside the peritoneal cavity, the child delivered by version, and the placenta removed. The peritoneal edges were sewn together, but the tissues did not unite well, as a fistula formed between the cervix and the abdominal wall. This

gradually closed, the patient making a good recovery. She was considered infected upon admission, as she had been repeatedly examined by those whose antiseptic precautions were not thorough. In operating upon this patient effort was made to close the peritoneum by the use of clamps, rendering the introduction of the uterine contents into the abdomen impossible. A second operation was made for contracted pelvis, and was an extraperitoneal cervical section. The bladder was moderately filled with 300 c.c. of water, and the uterus opened outside the peritoneal sac. The head of the child was delivered by the use of one blade of a pair of forceps. The child was large, asphyxiated, but readily revived. Incisions were then closed with catgut, and the abdomen closed. Both of these patients made good recoveries. In the discussion, Baumm stated his belief that it is a mistake to use the longitudinal incision if an effort is to be made to avoid opening the peritoneal cavity. In twenty-five such cases he was successful only twice in avoiding opening the peritoneum.

GYNECOLOGY.

UNDER THE CHARGE OF

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Malignant Disease of the Uterus; A Digest of 265 Cases Treated in the New Hospital for Women.—ANDERSON and PLATT (*Jour. Obst. and Gyn. Brit. Emp.*, 1908, iv, 381) report the work done in the New Hospital for Women, in London, from 1895 to 1907. There were 217 cases in which the disease affected the cervix, and 48 in which it affected the fundus. Hysterectomy was done by the abdominal route 90 times, with a mortality of 6, and 38 by the vaginal route, with 3 deaths. The vaginal route was employed principally previous to the year 1901, and the abdominal subsequently. Of the 217 cervix cases, 93 were squamous-celled carcinoma; 24 adenocarcinoma; 93 were unspecified; 2 were sarcoma; and 4 were endothelioma. Of the 48 fundus cases, 37 were adenocarcinoma, 1 was chorionepithelioma, and 10 were sarcoma. Cancer of the cervix was present in 29 of the 38 cases of vaginal hysterectomy, and the disease recurred in 12 in the vaginal scar within a few weeks from the time of operation. In 58 cases of abdominal hysterectomy for the cervical form of malignancy local recurrence occurred in 4 or possibly 5. Anderson and Platt insist that recurrence after the extended abdominal hysterectomy is in the iliac glands, and their removal is, therefore, strongly recommended. Of the 29 cases of cancer of the cervix subjected to vaginal hysterectomy, one patient is known to be well now, seven years after operation. The after-results in 58 abdominal hysterectomies for the same conditions are: 26 women are known to be alive and well, between one and one-half and four years after the operation; 17 developed symptoms of recurrence, 11 in the first year and 6 within

three and one-half years; 15 cannot be traced. Of the 48 patients suffering from malignant disease of the fundus, 39 were subjected to hysterectomy, 30 of them by the abdominal route. Of these, 22 have remained in perfect health two to seven years, 7 developed symptoms of recurrence from one to five years later, 3 died, and 7 cannot be traced. Out of the 37 cases of adenocarcinoma of the fundus, 17 had fibroids as well; and of 10 of sarcoma, 4 had fibroids. The vast proportion of women with cancer of the cervix are over thirty years of age, the average being forty-four and one-half years, and the average number of children was five. Six were unmarried and childless. In the fundus cases the average age was fifty-eight years, and the average number of children was 1; 18 of the 38 were unmarried and childless.

A Study of 131 Consecutive Cases of Fibroid Tumors of the Uterus Demanding Operation.—A MACCLAREN (*Annals of Surgery*, 1909, l, 281) reports the last 100 hysterectomies for uterine fibroids he has done and the myomectomies he has done during the same period of time, 31 in number; 78 per cent. of the hysterectomy patients were forty-five years of age or older, 1 was twenty-five years, 1 was twenty-eight years, and 28 per cent. were between thirty and forty-five years; 87 per cent. of the myomectomy patients were not more than forty years of age. In 3 of 60 myomectomies MacClaren has done fibroids have developed, requiring a second operation, in two of them hysterectomy. In the 3, the second operation was required within two years following the myomectomies. Myomectomy during pregnancy was done 4 times, with one miscarriage. MacClaren does not advise removal, during pregnancy, of a symptomless fibroid, nor abstaining from such operation for proper indications during pregnancy, even though the child be not viable. Three deaths occurred in the hysterectomy series and 2 in the myomecties. A very interesting statement is made regarding his gall-bladder surgery. In 20 consecutive cases of pelvic operations in which the gall-bladder had been opened and drained there had been 3 deaths, while the general mortality of his gall-bladder work in other cases was but 2 per cent. MacClaren has consequently abandoned Kelly's plan of removal of gallstones incidental to pelvic operations. Twice after myomectomy MacClaren has been obliged to do vaginal section for hematomas, although in the habit of employing vaginal drainage after this operation. In 10 per cent. of the hysterectomies this procedure was employed. Seventeen of the 100 hysterectomy patients had never married; 22 of the married ones had either miscarried or never been pregnant; 17 other married ones had not had a child for twenty years; 25 had had the last child from ten to twenty years before operation; 4 had given birth to children during the last preceding three years, and the remaining 11 of the hysterectomy subjects had each had a child between five and ten years before operation.

Rare Metastases of Uterine Carcinoma.—HEINRICH OFFERGELD (*Monatsschr. f. Geburtsh. u. Gynäk.*, 1909, xxix, 181) reviews the literature concerning rare metastatic growths from carcinoma of the uterus, and arrives at the following conclusions: In uterine carcinoma metastases occur very rarely in striated muscles; secondary growths occur with greater frequency in the myocardium. They occur in advanced cases

only, and arise hemogenetically without exception. The clinical symptoms of these metastases vary greatly, depending entirely on the function of the muscle affected (myocardium, eye muscles, muscles of the body; the latter not giving rise to any symptoms). On account of absence of all clinical symptoms the body musculature should be carefully examined at autopsy. The ureteral wall possesses a certain immunity against carcinoma; in two cases the carcinoma was inoperable, in the third case of this kind operation was performed successfully. Nothing definite is known of the mode of dissemination and of the clinical course. The apparently intact ureter should be examined microscopically at autopsy for possible microscopic carcinomatous infiltration of its wall. Carcinoma of the thoracic duct occurs more frequently in uterine cancer. The secondary carcinoma of the thoracic duct arises lymphogenetically through the medium of the inguinal, hypogastric, and lumbar plexuses, which empty into the thoracic duct. Carcinoma of the thoracic duct is of special importance, as it gives rise to further hematogenous metastases. Whether carcinoma of the thoracic duct in uterine cancer occurs in a large number of cases remains to be proved by future careful autopsies. Carcinoma of the supraclavicular and infraclavicular glands is found only in extensive glandular disease associated with uterine cancer. These metastases are of lymphogenous origin through the medium of the thoracic duct, with marked preference for the left side on account of the anatomical conditions. The mediastinal lymphatic glands are frequently found carcinomatous in uterine carcinoma, while secondary carcinoma of the mediastinum itself is very rare. The metastatic mediastinal tumors in uterine carcinoma arise lymphogenetically, either directly from the lymph glands, or by retrograde lymphogenous transportation.

The Treatment of Inoperable Cancer of the Uterus.—GELLHORN (*Amer. Jour. Obst.*, 1909, lix, 799) related to the Chicago Gynecological Society his technique of the application of acetone in the treatment of inoperable cancer of the uterus. While not claiming for this remedy curative properties, Gellhorn declares it the most efficient known in preventing hemorrhages and malodorous discharges. Two classes of cases are enumerated in which its application cannot satisfactorily be made. The first is composed of those *in extremis* when first seen. In these the body of the uterus seems to be merely a thin shell within which the ravages of the disease progress unhindered, even the most temporary amelioration being impossible, and the primary focus has lost all its significance. In the other class are placed cases in which the application of acetone would seem unpromising because of the attending unsurmountable technical difficulties. The principal difficulty is the unusual location of the cancer, as, for instance, the interior of the uterus, or the lower part of the vagina, or the vulva. This technical difficulty has brought about, as a substitute for liquid acetone, the white powder, acetone bisulphite, which can be used with a powder blower. It is less efficient than the liquid acetone.

DERMATOLOGY.

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A Case of Rodent Ulcer Cured by Radium.—SEQUEIRA (*Brit. Jour. Derm.*, February, 1909) reports a case of rodent ulcer which has been cured, and has remained so, five years before. The disease had begun at an unusually early period, when the patient, a young woman, was between fifteen and sixteen years old, and was situated upon the lower lid. The diagnosis of rodent ulcer was established by microscopic examination. The ulcer had partially healed under *x*-ray treatment, but this had to be stopped on account of conjunctivitis, and a relapse followed. In February, 1904, six applications of 10 milligrams of radium in tubes, having an estimated radio-activity of 500,000, were made on six successive days; this produced a marked reaction, and on February 24 the ulcer had healed.

Lupus Erythematosus Apparently Cured by Zinc Ionization.—MACLEOD (*Brit. Jour. Derm.*, March, 1909) reports a patient, a middle aged woman, having an erythematous lupus, of seven years' duration, in whom a cure had been obtained by the employment of zinc ionization. Two per cent. zinc sulphate was used, and a current of about 5 milliamperes, ten minutes at a time, to each patch. Nine sittings in all were given; and under this treatment the scaliness and redness had disappeared, leaving a pale, supple, slightly depressed scar.

GRAHAM LITTLE (*ibid.*) also reports a patient with erythematous lupus, a girl, aged fourteen years, in whom the same method of treatment had produced satisfactory results. He regards this treatment as the most rapid and efficient at our disposal in dealing with this obstinate affection.

The Treatment of Superficial Epithelioma by Curettage and Immediate Radiotherapy.—LENGLET and SOURDEAU (*Bull. de la soc. Française de derm. et de syphil.*, 1909, No. 2) have found the following method most successful in the treatment of superficial epithelioma: Every epithelioma, whatever may be its seat, is submitted, if possible, before irradiation, to complete, methodical scraping. If this cannot be done on account of its situation, and extirpation is possible, it is removed surgically. The scraping should be preceded by complete anesthesia of the entire area to be curetted; and if local anesthesia is not possible, recourse must be had to general anesthesia. The curettement must be most thoroughly done. Bleeding is usually stopped by simple compression,

but exceptionally the parts must be lightly touched with the actual cautery. Immediately after the operation, before the wound is dressed, it is irradiated, six H. being given, rarely seven, the rays used being No. 6 or No. 7, the latter being preferred. The second séance is not given until the time of reaction is largely passed, usually about the twentieth to the twentieth-eighth day; and the séances are thus continued until cicatrization occurs. The importance of this preliminary scraping is shown by the fact that Lenglet and Sourdeau have obtained a cure in 12 cases out of 36, with one irradiation, and in 9 with two séances. They believe that this combination of curettement and radiotherapy gives results superior to any other method of treatment.

Impetigo Contagiosa.—LEWANDOWSKY (*Archiv f. Derm. u. Syph.*, 1909, xciv, Heft 2 und 3) always found the streptococcus in 100 cases of impetigo examined bacteriologically, partly in pure culture, partly associated with the yellow staphylococcus, the former greatly exceeding the latter in numbers. In the great majority of cases of ecthyma the streptococcus was found in pure culture in the primary purulent lesion; in rare instances it was contaminated with isolated staphylococci. In one out of a small number of cases of pemphigus neonatorum and infantum examined, only staphylococci were found, while in the others streptococci in trifling numbers were also present. In impetiginous eczemas both streptococci and staphylococci were found, as well as in other serous lesions of various other dermatoses. Intra-epithelial inoculation with pure streptoccocus cultures produced impetigo contagiosa; and epithelial cutaneous inoculations were followed by ecthyma. Inoculations with staphylococci taken from impetigo were usually without result, or produced an abortive pustule. Staphylococci obtained from pus, eczema, impetigo, and some other diseases were affected in the same manner by agglutinating sera.

Occupation Carcinoma.—ZWEIG (*Derm. Ztschr.*, February, 1909) under the designation occupation carcinoma, reports three cases of carcinoma of the skin occurring in workmen employed in making coal briquettes. These briquettes are made by mixing ground coal with pitch and tar, subjecting the mixture to a high heat in an oven and afterward making it into bricks by pressure. Zweig thinks the injurious effects of this occupation upon the skin are due either to the dust arising in the process of mixing or to certain volatile substances produced in the heating of the mixture. This form of carcinoma is identical with those previously described as chimney-sweeper's cancer, paraffin cancer, etc. Zweig thinks there can be no doubt of the immediate relationship between this form of carcinoma and the chemically acting agents used in these occupations.

HYGIENE AND PUBLIC HEALTH.

UNDER THE CHARGE OF
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The Bleaching of Flour.—Within a few years the bleaching of flour has become a process of extensive employment in this country and Great Britain, possibly in other parts of the world also. When flour stands for some six weeks to as many months, it gradually becomes whiter. This change is attributed to "aging." The seller takes his samples and on the appearance of these, which may be many months old, he contracts to deliver so many carloads of the same kind. Recently ground flour shipped to fill this order disappoints the buyer, because it is not so white as the sample upon which he made his purchase. This has led to artificial bleaching with peroxide of nitrogen. The machinery for this purpose is of varied construction, but the principle is the same in all. The oil of the flour is bleached, and nitrite is produced. The chemist distinguishes between bleached and unbleached flour by the detection and estimation of nitrite in the former. The question of the bleaching of flour has been for some months before the Secretary of Agriculture at Washington, and some scientific work, with the object of determining the effect of the bleaching process upon the health of the consumer, has been done. In Halliburton's work (*Journal of Hygiene*, 1909, ix, 170) attention is given to the influence of nitrite on the digestive enzymes. All experiments were made in vitro. Nitrite, when present in the very small proportion of 1 to 32,000, markedly retards the digestion of starch by saliva. The time necessary to reach the achromatic point when tested with iodine was taken as the standard of measurement. The time required to reach this point in the presence of nitrite (1 to 32,000) was thirty-three minutes, while digestion in the control tube was complete at eighteen minutes. In testing proteolytic ferments the protein was stained with carmine and the tint of the solution used as a comparative measure. In tubes containing potassium nitrite (1 to 8000) reckoned as nitrite, digestion was wholly prevented and when the nitrite was reduced to 1 to 32,000 the digestive activity was only one-seventh of that in the tube free from nitrite. The question arose as to whether previous treatment with nitrite, even when the nitrite was removed before subjecting the protein to the action of the ferment, would affect digestion. For the purpose of solving this question three sets of tubes were prepared as follows: (1) Those with nitrite, (2) those previously treated with nitrite, and (3) those to which no nitrite had been added. The results were: In (1), digestion was slight; in (2), slight but greater than in (1); in (3), complete. Halliburton states his conclusions as follows: (1) The presence of nitrous acid (even in the comparatively innocuous form of a salt) hinders enzyme action. (2) Previous treatment with nitrous acid alters a protein in such a way as to render it less readily susceptible to the solvent

action of digestive juices. He also found that the starch in bleached flour is less easily digested by the saliva than that in unbleached flour. A like result followed in his experiment on the peptic digestion of the glutin in the flours.

(Halliburton's finding that nitrite so markedly impedes the salivary digestion of starch is, to say the least, unexpected, since nitrite is a normal constituent of human saliva, or, at least, has been long regarded as such by physiological chemists.—V. C. V.)

Sulphite in Cider and Perry.—DURHAM (*Ibid.*, 17) thinks that the permissible limit of the addition of this preservative to these beverages should be expressed as "total sulphur dioxide" obtained by distillation with phosphoric acid, and that this should not exceed 100 mg. per liter. A trace should be defined as less than 10 (possibly 20) mg. per liter, and when this is exceeded, declaration of the presence of sulphite should be made on the label. All cider makers who desire to use or who do use preservatives should be registered and under official inspection.

Chronic Lead Poisoning.—Notwithstanding recent sanitary improvements in manufacturing establishments, chronic lead poisoning is by no means infrequent in large white lead factories. Animals about such factories vary greatly in susceptibility to the poison. Apparently it is without effect upon rats, which may infest the white lead factory in great numbers, but cats brought in to drive out these pests soon succumb to chronic lead poisoning. There has been more or less discussion as to whether or not poisoning may and does result from the inhalation of the dust that always permeates the atmosphere of white lead factories especially of those not adequately supplied with fan exhausts. GOADBY (*Jour. of Hygiene*, 1909, ix, 122) has investigated this question experimentally, using cats, and has answered the questions in the affirmative. The material used consisted of: (1) Flue dust, containing from 50 to 60 per cent. of lead oxide. In the process of desilverizing, zinc is added to molten lead containing gold and silver; the zinc holds the precious metals, and is then separated from the lead by differences in melting point. The lead from which the zinc has extracted the gold and silver, known as "poor lead," is run into a pot and there treated with air and steam under pressure, for the purpose of ozidizing any zinc that it may contain, but some of the lead also is oxidized, and in the form of a free dust finds its way to the workmen. (2) Litharge is broken into large lumps by hand, and then these are ground in a mill. Some finely divided litharge reaches the men who feed the mill. (3) More or less white lead in the form of a fine dust escapes from the ducts leading from the machines used in packing dry white lead in barrels.

The first effect noticed in the cats caused to inhale these dusts was an alteration in the face due to the absorption of the orbital and buccinator fat, and giving the animal a pinched appearance much like that shown by men poisoned in lead factories. Colic was the next symptom. The cats were obstinately constipated, and showed much abdominal distress. Then the animals developed muscular weakness, shown

especially in the extensors of the limbs, giving a curious stiff gait. The poisoned cat walks on the tip of its toes, and in endeavoring to turn around is compelled to arch its back and draw its feet close together to prevent falling over. The back muscles are distinctly weak, so much so that when the animal is held up by placing the forefinger and thumb behind the ears it hangs down straight and can make no move to twist its body or claw the hand. When it jumps off a table it falls upon its belly, the extensor muscles being unequal to the strain. At first the loss of weight was progressive, but, after falling to a certain point, it remained constant, in some cases showing a tendency to rise later. The experiments were not pushed to a fatal termination, but the animals were killed. No blood examinations were made.

Transmission of Microörganisms through Berkefeld Filters.—BULLOCK and CRAW (*Ibid.*, 35) conclude their study of this point with the following findings: (1) Of two filters with porcelain nipples, one gave a non-sterile filtrate with London tap water immediately, and the other after three hours and forty minutes. These filters had been boiled for one hour but not autoclaved. (2) Of three with metal nipples, two gave immediate contamination with tap water, and the third after two hours and forty minutes. These filters also were new and had only been boiled for one hour. (3) Two dried Berkefeld filters, one with metal and one with porcelain nipples, on immersion of a portion of their walls in a water culture of Bacillus prodigiosus, allowed this organism to pass into the anterior of the filters.

Antiplague Measures in San Francisco.—BLUE (*Ibid.*, 1) sums up the measures that have apparently proved satisfactory in the eradication of the plague at San Francisco as follows: (1) Attack upon the habitation and food supply of the rat; (2) destruction of rat burrows and nests; (3) prevention of access of rats to food by concreting stables, warehouses, markets, etc.; (4) exclusion of rats from houses by the use of concrete or other impervious material; (5) filling of rat burrows with strong solution of chloride of lime; (6) disinfection of all places in which either human or rodent cases have been detected.

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